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TERMINAL (ENTER 1, 2, 3, OR ?):2

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NEWS 1 Web Page URLs for STN Seminar Schedule - N. America  
NEWS 2 "Ask CAS" for self-help around the clock  
NEWS 3 SEP 09 CA/CAPLUS records now contain indexing from 1907 to the  
present  
NEWS 4 AUG 05 New pricing for EUROPATFULL and PCTFULL effective  
August 1, 2003  
NEWS 5 AUG 13 Field Availability (/FA) field enhanced in BEILSTEIN  
NEWS 6 AUG 18 Data available for download as a PDF in RDISCLOSURE  
NEWS 7 AUG 18 Simultaneous left and right truncation added to PASCAL  
NEWS 8 AUG 18 FROSTI and KOSMET enhanced with Simultaneous Left and Right  
Truncation  
NEWS 9 AUG 18 Simultaneous left and right truncation added to ANABSTR  
NEWS 10 SEP 22 DIPPR file reloaded  
NEWS 11 SEP 25 INPADOC: Legal Status data to be reloaded  
NEWS 12 SEP 29 DISSABS now available on STN  
NEWS 13 OCT 10 PCTFULL: Two new display fields added  
NEWS 14 OCT 21 BIOSIS file reloaded and enhanced  
  
NEWS EXPRESS OCTOBER 01 CURRENT WINDOWS VERSION IS V6.01a, CURRENT  
MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),  
AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003  
NEWS HOURS STN Operating Hours Plus Help Desk Availability  
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NEWS LOGIN Welcome Banner and News Items  
NEWS PHONE Direct Dial and Telecommunication Network Access to STN  
NEWS WWW CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that  
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\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 12:37:13 ON 27 OCT 2003

=> file medline, uspatful, dgene, embase, wpids, fsta, jicst, biosis, biobusiness		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 12:37:37 ON 27 OCT 2003

FILE 'USPATFULL' ENTERED AT 12:37:37 ON 27 OCT 2003

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NEWS 3 SEP 09 CA/CAPLUS records now contain indexing from 1907 to the  
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NEWS 4 AUG 05 New pricing for EUROPATFULL and PCTFULL effective  
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NEWS 8 AUG 18 FROSTI and KOSMET enhanced with Simultaneous Left and Right  
Truncation  
NEWS 9 AUG 18 Simultaneous left and right truncation added to ANABSTR  
NEWS 10 SEP 22 DIPPR file reloaded  
NEWS 11 SEP 25 INPADOC: Legal Status data to be reloaded  
NEWS 12 SEP 29 DISSABS now available on STN  
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NEWS 14 OCT 21 BIOSIS file reloaded and enhanced  
  
NEWS EXPRESS OCTOBER 01 CURRENT WINDOWS VERSION IS V6.01a, CURRENT  
MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),  
AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003  
NEWS HOURS STN Operating Hours Plus Help Desk Availability  
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NEWS LOGIN Welcome Banner and News Items  
NEWS PHONE Direct Dial and Telecommunication Network Access to STN  
NEWS WWW CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that  
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result in loss of user privileges and other penalties.

\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 12:59:28 ON 27 OCT 2003

=> file medline, uspatful, dgene, embase, wpids, fsta, biosis,		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 12:59:46 ON 27 OCT 2003

FILE 'USPATFULL' ENTERED AT 12:59:46 ON 27 OCT 2003

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FILE 'WPIDS' ENTERED AT 12:59:46 ON 27 OCT 2003  
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FILE 'FSTA' ENTERED AT 12:59:46 ON 27 OCT 2003  
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FILE 'BIOSIS' ENTERED AT 12:59:46 ON 27 OCT 2003  
COPYRIGHT (C) 2003 BIOLOGICAL ABSTRACTS INC.(R)

=> e ballance, d/au

E1	1	BALLANCE WILLIAM A JR/AU
E2	5	BALLANCE X/AU
E3	0 -->	BALLANCE, D/AU
E4	1	BALLANCIEN B/AU
E5	21	BALLANCO G A/AU
E6	4	BALLANCO GERARD A/AU
E7	1	BALLANCO JULIUS/AU
E8	1	BALLANCO M F/AU
E9	1	BALLANCO MICHAEL F/AU
E10	1	BALLAND/AU
E11	58	BALLAND A/AU
E12	6	BALLAND ALAIN/AU

=> e sleep,d/au

E1	6	SLEEP TAMSIN J/AU
E2	1	SLEEP W/AU
E3	0 -->	SLEEP, D/AU
E4	3	SLEEPER A A/AU
E5	1	SLEEPER A B/AU
E6	7	SLEEPER A M/AU
E7	1	SLEEPER AMANDA A/AU
E8	1	SLEEPER B/AU
E9	10	SLEEPER B P/AU
E10	5	SLEEPER C A/AU
E11	2	SLEEPER D A/AU
E12	1	SLEEPER DAVID ALLEN/AU

=> e prior,c/au

E1	2	PRIOR YVONNE/AU
E2	1	PRIOR ZAK G R/AU
E3	0 -->	PRIOR, C/AU
E4	15	PRIORA C/AU
E5	1	PRIORA FABIO/AU
E6	1	PRIORA GIUSEPPE/AU
E7	2	PRIORA P/AU
E8	25	PRIORA P M/AU
E9	1	PRIORA RITA/AU
E10	1	PRIORA S A/AU
E11	5	PRIORA U/AU
E12	1	PRIORDE CASTRO C/AU

=> e sadeghi,h/au

E1	8	SADEGHI ZADEH M/AU
E2	2	SADEGHI ZADEH MAJID/AU
E3	0 -->	SADEGHI, H/AU
E4	2	SADEGHIAN A/AU

E5	1	SADEGHIAN ALI/AU
E6	13	SADEGHIAN E/AU
E7	5	SADEGHIAN H/AU
E8	2	SADEGHIAN HAMID/AU
E9	32	SADEGHIAN K/AU
E10	1	SADEGHIAN K S/AU
E11	2	SADEGHIAN KEN/AU
E12	8	SADEGHIAN KENNETH/AU

AB Nucleic acid encoding a functional HTLV-III/LAV (HIV-1) protein having trans-activating ability, and expression vectors comprising this nucleic acid are described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 1998:104629 USPATFULL  
TITLE: Nucleic acid encoding HIV-1 tat protein  
INVENTOR(S): Haseltine, William Alan, Cambridge, MA,  
United States  
Rosen, Craig A., Brookline, MA, United States  
Sodroski, Joseph Gerald, Cambridge, MA, United States  
Wong-Staal, Flossie, San Diego, CA, United States  
Arya, Suresh K., Gaithersburg, MD, United States  
PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Boston, MA, United States  
(U.S. corporation)  
The United States of America as represented by the  
Department of Health and Human Services, Washington,  
DC, United States (U.S. government)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5801056		19980901
APPLICATION INFO.:	US 1993-131898		19931005 (8)
RELATED APPLN. INFO.:	Division of Ser. No. US 1992-869053, filed on 14 Apr 1992, now abandoned And a continuation-in-part of Ser. No. US 1988-172152, filed on 23 Mar 1988, now abandoned which is a continuation-in-part of Ser. No. US 1985-780925, filed on 27 Sep 1985, now abandoned , said Ser. No. US -869053 which is a continuation of Ser. No. US 1990-604607, filed on 26 Oct 1990, now abandoned which is a division of Ser. No. US 1985-806263, filed on 6 Dec 1985, now patented, Pat. No. US 4981790		

	NUMBER	DATE
PRIORITY INFORMATION:	CA 1985-482374	19850524
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	Granted	
PRIMARY EXAMINER:	Fleisher, Mindy	
ASSISTANT EXAMINER:	Railey, II, Johnny F.	
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I. Dike, Bronstein, Roberts & Cushman, LLP	
NUMBER OF CLAIMS:	13	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	14 Drawing Figure(s); 7 Drawing Page(s)	
LINE COUNT:	855	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L1 ANSWER 2 OF 2 USPATFULL on STN  
TI Assay methods for tat cell lines  
AB Assays screened for compounds that inhibit tat transactivation of the HIV (HTLV-III) LTR are taught. The assay involves tranfecting a cell line containing the tat gene by a vector containing a gene under the control of an HIV-1 LTR, adding the compound to be screened and determining the effect of the compound by looking at the effect of tat as measured by the expression of the gene under the control of the HIV LTR.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 1998:104559 USPATFULL  
TITLE: Assay methods for tat cell lines  
INVENTOR(S): Haseltine, William Alan, Cambridge, MA,  
United States  
Rosen, Craig A., Brookline, MA, United States

PATENT ASSIGNEE(S) : Sodroski, Joseph Gerald, Cambridge, MA, United States  
Goh, Wei Chun, Somerville, MA, United States  
Dana Farber Cancer Institute, Boston, MA, United States  
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5800986		19980901
APPLICATION INFO.:	US 1995-456346		19950601 (8)
RELATED APPLN. INFO.:	Division of Ser. No. US 1994-213368, filed on 14 Mar 1994, now abandoned which is a continuation of Ser. No. US 1992-869053, filed on 14 Apr 1992, now abandoned which is a continuation of Ser. No. US 1990-604607, filed on 26 Oct 1990, now abandoned which is a division of Ser. No. US 1985-806263, filed on 6 Dec 1985, now patented, Pat. No. US 4981790 which is a continuation-in-part of Ser. No. US 1984-614297, filed on 25 May 1984, now patented, Pat. No. US 4738922		

	NUMBER	DATE
PRIORITY INFORMATION:	CA 1985-432374	19850524
	WO 1985-US985	19850524
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	Granted	
PRIMARY EXAMINER:	Elliott, George C.	
ASSISTANT EXAMINER:	McKelvey, Terry A.	
LEGAL REPRESENTATIVE:	Conlin, David C., Eisenstein, Ronald I. Dike, Bronstein, Roberts & Cushman, LLP	
NUMBER OF CLAIMS:	8	
EXEMPLARY CLAIM:	8	
NUMBER OF DRAWINGS:	14 Drawing Figure(s); 7 Drawing Page(s)	
LINE COUNT:	871	
CAS INDEXING IS AVAILABLE FOR THIS PATENT.		

=> dhis  
DHIS IS NOT A RECOGNIZED COMMAND  
The previous command name entered was not recognized by the system.  
For a list of commands available to you in the current file, enter  
"HELP COMMANDS" at an arrow prompt (=>).

=> d his

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FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, JICST-EPLUS,  
BIOSIS, BIOBUSINESS' ENTERED AT 12:37:37 ON 27 OCT 2003

E ROSEN, C/AU

E HASELTINE, W/AU

L1 2 S E2  
L2 78 S E1  
L3 4 S FUSION ALBUMIN PROTEIN

=> s l2 and l3

L4 0 L2 AND L3

=> d l3 ti abs ibib tot

L3 ANSWER 1 OF 4 USPATFULL on STN

TI Compositions and methods for the therapy and diagnosis of colon cancer  
AB Compositions and methods for the therapy and diagnosis of cancer,  
particularly colon cancer, are disclosed. Illustrative compositions  
comprise one or more colon tumor polypeptides, immunogenic portions

thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly colon cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:237907 USPATFULL  
TITLE: Compositions and methods for the therapy and diagnosis of colon cancer  
INVENTOR(S): King, Gordon E., Shoreline, WA, UNITED STATES  
Meagher, Madeleine Joy, Seattle, WA, UNITED STATES  
Xu, Jiangchun, Bellevue, WA, UNITED STATES  
Secrist, Heather, Seattle, WA, UNITED STATES  
Jiang, Yuqiu, Kent, WA, UNITED STATES  
PATENT ASSIGNEE(S): Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003166064	A1	20030904
APPLICATION INFO.:	US 2002-99926	A1	20020314 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2001-33528, filed on 26 Dec 2001, PENDING Continuation-in-part of Ser. No. US 2001-920300, filed on 31 Jul 2001, PENDING		

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-302051P	20010629 (60)
	US 2001-279763P	20010328 (60)
	US 2000-223283P	20000803 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092	
NUMBER OF CLAIMS:	17	
EXEMPLARY CLAIM:	1	
LINE COUNT:	8531	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 2 OF 4 USPATFULL on STN

TI Compositions and methods for the therapy and diagnosis of pancreatic cancer

AB Compositions and methods for the therapy and diagnosis of cancer, particularly pancreatic cancer, are disclosed. Illustrative compositions comprise one or more pancreatic tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly pancreatic cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:106233 USPATFULL  
TITLE: Compositions and methods for the therapy and diagnosis of pancreatic cancer  
INVENTOR(S): Benson, Darin R., Seattle, WA, UNITED STATES  
Kalos, Michael D., Seattle, WA, UNITED STATES  
Lodes, Michael J., Seattle, WA, UNITED STATES  
Persing, David H., Redmond, WA, UNITED STATES  
Hepler, William T., Seattle, WA, UNITED STATES  
Jiang, Yuqiu, Kent, WA, UNITED STATES  
PATENT ASSIGNEE(S): Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003073144	A1	20030417	
APPLICATION INFO.:	US 2002-60036	A1	20020130	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-333626P	20011127 (60)
	US 2001-305484P	20010712 (60)
	US 2001-265305P	20010130 (60)
	US 2001-267568P	20010209 (60)
	US 2001-313999P	20010820 (60)
	US 2001-291631P	20010516 (60)
	US 2001-287112P	20010428 (60)
	US 2001-278651P	20010321 (60)
	US 2001-265682P	20010131 (60)

DOCUMENT TYPE: Utility  
FILE SEGMENT: APPLICATION  
LEGAL REPRESENTATIVE: SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092  
NUMBER OF CLAIMS: 17  
EXEMPLARY CLAIM: 1  
LINE COUNT: 14253  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 3 OF 4 USPATFULL on STN  
TI Compositions and methods for the therapy and diagnosis of colon cancer  
AB Compositions and methods for the therapy and diagnosis of cancer, particularly colon cancer, are disclosed. Illustrative compositions comprise one or more colon tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly colon cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
ACCESSION NUMBER: 2002:272801 USPATFULL  
TITLE: Compositions and methods for the therapy and diagnosis of colon cancer  
INVENTOR(S): Stolk, John A., Bothell, WA, UNITED STATES  
Xu, Jiangchun, Bellevue, WA, UNITED STATES  
Chenault, Ruth A., Seattle, WA, UNITED STATES  
Meagher, Madeleine Joy, Seattle, WA, UNITED STATES  
PATENT ASSIGNEE(S): Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002150922	A1	20021017	
APPLICATION INFO.:	US 2001-998598	A1	20011116	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-304037P	20010710 (60)
	US 2001-279670P	20010328 (60)
	US 2001-267011P	20010206 (60)
	US 2000-252222P	20001120 (60)

DOCUMENT TYPE: Utility  
FILE SEGMENT: APPLICATION  
LEGAL REPRESENTATIVE: SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092  
NUMBER OF CLAIMS: 17



EXEMPLARY CLAIM: 1  
LINE COUNT: 9233  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 4 OF 4 USPATFULL on STN

TI Compositions and methods for the therapy and diagnosis of ovarian cancer  
AB Compositions and methods for the therapy and diagnosis of cancer, particularly ovarian cancer, are disclosed. Illustrative compositions comprise one or more ovarian tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly ovarian cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:243051 USPATFULL  
TITLE: Compositions and methods for the therapy and diagnosis of ovarian cancer  
INVENTOR(S): Algate, Paul A., Issaquah, WA, UNITED STATES  
Jones, Robert, Seattle, WA, UNITED STATES  
Harlocker, Susan L., Seattle, WA, UNITED STATES  
PATENT ASSIGNEE(S): Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002132237	A1	20020919
APPLICATION INFO.:	US 2001-867701	A1	20010529 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-207484P	20000526 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092	
NUMBER OF CLAIMS:	11	
EXEMPLARY CLAIM:	1	
LINE COUNT:	25718	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> s 12 and albumin protein  
L5 1 L2 AND ALBUMIN PROTEIN

=> d 15 ti abs ibib tot

L5 ANSWER 1 OF 1 USPATFULL on STN

TI Albumin fusion proteins  
AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disorders or conditions using albumin fusion proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:181414 USPATFULL  
TITLE: Albumin fusion proteins

INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Haseltine, William A., Washington, DC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003125247	A1	20030703
APPLICATION INFO.:	US 2001-833041	A1	20010412 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-256931P	20001221 (60)
	US 2000-199384P	20000425 (60)
	US 2000-229358P	20000412 (60)

DOCUMENT TYPE: Utility  
FILE SEGMENT: APPLICATION  
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE,  
ROCKVILLE, MD, 20850  
NUMBER OF CLAIMS: 29  
EXEMPLARY CLAIM: 1  
NUMBER OF DRAWINGS: 20 Drawing Page(s)  
LINE COUNT: 15235  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d his

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FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, JICST-EPLUS,  
BIOSIS, BIOBUSINESS' ENTERED AT 12:37:37 ON 27 OCT 2003

E ROSEN, C/AU  
E HASELTINE, W/AU

L1 2 S E2  
L2 78 S E1  
L3 4 S FUSION ALBUMIN PROTEIN  
L4 0 S L2 AND L3  
L5 1 S L2 AND ALBUMIN PROTEIN

=> d l2 ti abs ibib tot

L2 ANSWER 1 OF 78 USPATFULL on STN  
TI Human DNA mismatch repair proteins  
AB The present invention discloses three human DNA repair proteins and DNA  
(RNA) encoding such proteins and a procedure for producing such proteins  
by recombinant techniques. One of the human DNA repair proteins, hMLH1,  
has been mapped to chromosome 3 while hMLH2 has been mapped to  
chromosome 2 and hMLH3 has been mapped to chromosome 7. The  
polynucleotide sequences of the DNA repair proteins may be used for  
therapeutic and diagnostic treatments of a hereditary susceptibility to  
cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:228227 USPATFULL  
TITLE: Human DNA mismatch repair proteins  
INVENTOR(S): Haseltine, William A., Washington, DC, United States  
Ruben, Steven M., Brookeville, MD, United States  
Wei, Ying-Fei, Berkeley, CA, United States  
Adams, Mark D., Rockville, MD, United States  
Fleischmann, Robert D., Gaithersburg, MD, United States  
Fraser, Claire M., Potomac, MD, United States  
Fuldner, Rebecca A., Barnesville, MD, United States  
Kirkness, Ewen F., Olney, MD, United States

PATENT ASSIGNEE(S): Rosen, Craig A., Laytonsville, MD, United States  
 Vogelstein, Bert, Baltimore, MD, United States  
 Kinzler, Kenneth W., Bel Air, MD, United States  
 Nicolaides, Nicholas C., Boothwyn, PA, United States  
 Papadopoulos, Nickolas, Brookline, MA, United States  
 Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)  
 The Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6610477	B1	20030826
APPLICATION INFO.:	US 1995-465679		19950606 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1994-294312, filed on 23 Aug 1994, now patented, Pat. No. US 6380369 Continuation-in-part of Ser. No. US 1994-210143, filed on 16 Mar 1994 Continuation-in-part of Ser. No. US 1994-187757, filed on 27 Jan 1994		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Horlick, Kenneth R.		
LEGAL REPRESENTATIVE:	Human Genome Sciences, Inc.		
NUMBER OF CLAIMS:	13		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	27 Drawing Figure(s); 26 Drawing Page(s)		
LINE COUNT:	2655		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.			

L2 ANSWER 2 OF 78 USPATFULL on STN  
 TI Albumin fusion proteins  
 AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disorders or conditions using albumin fusion proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
 ACCESSION NUMBER: 2003:181414 USPATFULL  
 TITLE: Albumin fusion proteins  
 INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES  
 Haseltine, William A., Washington, DC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003125247	A1	20030703
APPLICATION INFO.:	US 2001-833041	A1	20010412 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-256931P	20001221 (60)
	US 2000-199384P	20000425 (60)
	US 2000-229358P	20000412 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850	
NUMBER OF CLAIMS:	29	

EXEMPLARY CLAIM: 1  
NUMBER OF DRAWINGS: 20 Drawing Page(s)  
LINE COUNT: 15235  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 3 OF 78 USPATFULL on STN  
TI HUMAN DNA MISMATCH REPAIR PROTEIN  
AB The present invention discloses three human DNA repair proteins and DNA (RNA) encoding such proteins. The DNA repair proteins may be produced by recombinant DNA techniques. One of the human DNA repair proteins, hmlh1, has been mapped on chromosome 3. The polynucleotide sequences of DNA repair proteins may be used for diagnosis of a hereditary susceptibility to cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:127014 USPATFULL  
TITLE: HUMAN DNA MISMATCH REPAIR PROTEIN  
INVENTOR(S): **HASELTINE, WILLIAM A.**, WASHINGTON, DC, UNITED STATES  
RUBEN, STEVEN, OLNEY, MD, UNITED STATES  
WEI, YING-FEI, DARNESTOWN, MD, UNITED STATES  
ADAMS, MARK D., NORTH POTOMAC, MD, UNITED STATES  
FLEISCHMANN, ROBERT D., WASHINGTON, DC, UNITED STATES  
FRASER, CLAIRE M., QUEENSTOWN, MD, UNITED STATES  
ROSEN, CRAIG A., LAYTONSVILLE, MD, UNITED STATES  
FULDNER, REBECCA A., BARNESVILLE, MD, UNITED STATES  
KIRKNESS, EWEN F., WASHINGTON, DC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003087226	A1	20030508
	US 6620619	B2	20030916
APPLICATION INFO.:	US 1994-210143	A1	19940316 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1994-187757, filed on 27 Jan 1994, GRANTED, Pat. No. US 6482606		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850		
NUMBER OF CLAIMS:	19		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	6 Drawing Page(s)		
LINE COUNT:	1017		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 4 OF 78 USPATFULL on STN  
TI Human DNA mismatch repair proteins  
AB The present invention discloses three human DNA repair proteins and DNA (RNA) encoding such proteins and a prodeudre for producing such proteins by recombinant techniques. One of the human DNA repair proteins, hMLH1, has been mapped to chromosome 3 while hMLH2 has been mapped to chromosome 2 and hMLH3 has been mapped to chromosome 7. The polynucleotide sequences of the DNA repair proteins may be used for therapeutic and diagnostic treatments of a hereditary susceptibility to cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:37532 USPATFULL  
TITLE: Human DNA mismatch repair proteins  
INVENTOR(S): **Has ltine, William A.**, Washington, DC, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
Wei, Ying-Fei, Berkeley, CA, UNITED STATES  
Adams, Mark D., Rockville, MD, UNITED STATES

Fleischmann, Robert D., Gaithersburg, MD, UNITED STATES  
 Fraser, Claire M., Potomac, MD, UNITED STATES  
 Fuldner, Rebecca A., Barnesville, MD, UNITED STATES  
 Kirkness, Ewen F., Olney, MD, UNITED STATES  
 Rosen, Craig A., Laytonsville, MD, UNITED STATES  
 PATENT ASSIGNEE(S): Human Genome Sciences, Inc., Rockville, MD (U.S.  
 corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003027177	A1	20030206
APPLICATION INFO.:	US 2002-79429	A1	20020222 (10)
RELATED APPLN. INFO.:	Division of Ser. No. US 1995-468024, filed on 6 Jun 1995, PENDING Continuation-in-part of Ser. No. WO 1995-US1035, filed on 25 Jan 1995, UNKNOWN Continuation-in-part of Ser. No. US 1994-294312, filed on 23 Aug 1994, GRANTED, Pat. No. US 6380369 Continuation-in-part of Ser. No. US 1994-210143, filed on 16 Mar 1994, PENDING Continuation-in-part of Ser. No. US 1994-187757, filed on 27 Jan 1994, PENDING Division of Ser. No. US 1995-465679, filed on 6 Jun 1995, PENDING Continuation-in-part of Ser. No. US 1994-294312, filed on 23 Aug 1994, GRANTED, Pat. No. US 6380369 Continuation-in-part of Ser. No. US 1994-210143, filed on 16 Mar 1994, PENDING Continuation-in-part of Ser. No. US 1994-187757, filed on 27 Jan 1994, PENDING Continuation-in-part of Ser. No. US 1994-294312, filed on 23 Aug 1994, GRANTED, Pat. No. US 6380369 Continuation-in-part of Ser. No. US 1994-210143, filed on 16 Mar 1994, PENDING Continuation-in-part of Ser. No. US 1994-187757, filed on 27 Jan 1994, PENDING Continuation-in-part of Ser. No. US 1994-210143, filed on 16 Mar 1994, PENDING Continuation-in-part of Ser. No. US 1994-187757, filed on 27 Jan 1994, PENDING Continuation-in-part of Ser. No. US 1994-187757, filed on 27 Jan 1994, PENDING		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850		
NUMBER OF CLAIMS:	14		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	26 Drawing Page(s)		
LINE COUNT:	2724		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.			

L2 ANSWER 5 OF 78 USPATFULL on STN  
 TI Human DNA mismatch repair polynucleotides  
 AB The present invention discloses three human DNA repair proteins and DNA  
 (RNA) encoding such proteins. The DNA repair proteins may be produced by  
 recombinant DNA techniques. One of the human DNA repair proteins, hmlh1,  
 has been mapped on chromosome 3. The polynucleotide sequences of DNA  
 repair proteins may be used for diagnosis of a hereditary susceptibility  
 to cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
 ACCESSION NUMBER: 2002:303858 USPATFULL  
 TITLE: Human DNA mismatch repair polynucleotides  
 INVENTOR(S): Adams, Mark D., North Potomac, MD, United States  
 Fleischmann, Robert D., Washington, DC, United States  
 Fraser, Claire M., Queenstown, MD, United States  
 Fuldner, Rebecca A., Barnesville, MD, United States  
 Kirkness, Ewen F., Washington, DC, United States  
 Haseltine, William A., Washington, DC, United

States

Rosen, Craig A., Laytonsville, MD, United States

Ruben, Steve, Olney, MD, United States

Wei, Ying-Fei, Darnestown, MD, United States

PATENT ASSIGNEE(S): Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6482606	B1	20021119
APPLICATION INFO.:	US 1994-187757		19940127 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	McKelvey, Terry		
LEGAL REPRESENTATIVE:	Human Genome Sciences, Inc.		
NUMBER OF CLAIMS:	26		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	8 Drawing Figure(s); 8 Drawing Page(s)		
LINE COUNT:	1290		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 6 OF 78 USPATFULL on STN

TI Human DNA Ligase IV

AB A human DNA Ligase IV polypeptide and DNA (RNA) encoding such polypeptide and a procedure for producing such polypeptide by recombinant techniques is disclosed. Also disclosed are methods for utilizing such polypeptide via gene therapy for the treatment of disorders associated with a defect in DNA Ligase IV. Antagonists against such polypeptides and their use as a therapeutic to destroy unwanted cells are also disclosed. Diagnostic assays to detect mutant DNA Ligase IV genes are also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:246554 USPATFULL

TITLE: Human DNA Ligase IV

INVENTOR(S): Wei, Ying-Fei, Darnestown, MD, United States  
**Haseltine, William A.**, Washington, DC, United States

PATENT ASSIGNEE(S): Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6455274	B1	20020924
APPLICATION INFO.:	US 1995-461562		19950605 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. WO 1994-US12922, filed on 8 Nov 1994		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Pak, Michael		
LEGAL REPRESENTATIVE:	Human Genome Sciences, Inc.		
NUMBER OF CLAIMS:	46		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	15 Drawing Figure(s); 15 Drawing Page(s)		
LINE COUNT:	1792		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 7 OF 78 USPATFULL on STN

TI Human DNA Ligase IV

AB A human DNA Ligase IV polypeptide and DNA (RNA) encoding such polypeptide and a procedure for producing such polypeptide by recombinant techniques is disclosed. Also disclosed are methods for utilizing such polypeptide via gene therapy for the treatment of disorders associated with a defect in DNA Ligase IV. Antagonists against

such polypeptides and their use as a therapeutic to destroy unwanted cells are also disclosed. Diagnostic assays to detect mutant DNA Ligase IV genes are also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:243145 USPATFULL  
TITLE: Human DNA Ligase IV  
INVENTOR(S): Wei, Ying-Fei, Berkeley, CA, UNITED STATES  
**Haseltine, William A.**, Washington, DC, UNITED STATES  
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., Rockville, MD, UNITED STATES, 20850 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002132331	A1	20020919
APPLICATION INFO.:	US 2002-141132	A1	20020509 (10)
RELATED APPLN. INFO.:	Division of Ser. No. US 1995-461562, filed on 5 Jun 1995, PENDING Continuation-in-part of Ser. No. WO 1994-US12922, filed on 8 Nov 1994, UNKNOWN		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850		
NUMBER OF CLAIMS:	20		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	15 Drawing Page(s)		
LINE COUNT:	1669		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 8 OF 78 USPATFULL on STN

TI Human genes, sequences and expression products-16  
AB A DNA sequence of SEQ ID NOS:1-12483. An isolated DNA sequence containing the coding region of a human gene and a DNA sequence identified in SEQ ID NOS:1-12483. An isolated DNA sequence containing the coding region of a human gene that contains a DNA sequence present in ATCC Deposit No. 75916. A DNA sequence hybridizable with a DNA sequence of SEQ ID NOS:1-12483 and isolatable from other DNA in ATCC Deposit No. 75916. Expression vectors containing any of the above. Proteins expressed from any of the above.

ACCESSION NUMBER: 2002:206157 USPATFULL  
TITLE: Human genes, sequences and expression products-16  
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
Dillon, Patrick J., Gaithersburg, MD, UNITED STATES  
Li, Haodong, Gaithersburg, MD, UNITED STATES  
**Haseltine, William A.**, Washington, DC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002110850	A1	20020815
APPLICATION INFO.:	US 2001-783590	A1	20010215 (9)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1995-420856, filed on 12 Apr 1995, ABANDONED		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850		
NUMBER OF CLAIMS:	27		
EXEMPLARY CLAIM:	1		
LINE COUNT:	2546		

L2 ANSWER 9 OF 78 USPATFULL on STN

TI Human DNA mismatch repair proteins

AB The invention discloses three human DNA repair proteins and DNA (RNA) encoding such proteins and a procedure for producing such proteins by recombinant techniques. One of the human DNA repair proteins, hMLH1, has been mapped to chromosome 3 while hMLH2 has been mapped to chromosome 2 and hMLH3 has been mapped to chromosome 7. The polynucleotide sequences of the DNA repair proteins may be used for therapeutic and diagnostic treatments of a hereditary susceptibility to cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:168073 USPATFULL

TITLE: Human DNA mismatch repair proteins

INVENTOR(S): Haseltine, William A., Washington, DC, United States

Ruben, Steven M., Olney, MD, United States

Wei, Ying-Fei, Darnestown, MD, United States

Adams, Mark D., North Potomac, MD, United States

Fleischmann, Robert D., Gaithersburg, MD, United States

Fraser, Claire M., Potomac, MD, United States

Fuldner, Rebecca A., Barnesville, MD, United States

Kirkness, Ewen F., Olney, MD, United States

Rosen, Craig A., Laytonsville, MD, United States

PATENT ASSIGNEE(S): Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
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PATENT INFORMATION:	US 6416984	B1	20020709
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APPLICATION INFO.:	US 1995-468024		19950606 (8)
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RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. WO 1995-US1035, filed on 25 Jan 1995 Continuation-in-part of Ser. No. US 1994-294312, filed on 23 Aug 1994 Continuation-in-part of Ser. No. US 1994-210143, filed on 16 Mar 1994 Continuation-in-part of Ser. No. US 1994-187757, filed on 27 Jan 1994		
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DOCUMENT TYPE: Utility

FILE SEGMENT: GRANTED

PRIMARY EXAMINER: Nashed, Nashaat T.

LEGAL REPRESENTATIVE: Human Genome Sciences, Inc.

NUMBER OF CLAIMS: 48

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 28 Drawing Figure(s); 26 Drawing Page(s)

LINE COUNT: 2754

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 10 OF 78 USPATFULL on STN

TI Human DNA mismatch repair proteins

AB The present invention discloses three human DNA repair proteins and DNA (RNA) encoding such proteins. The DNA repair proteins which may be produced by recombinant DNA techniques. One of the human DNA repair proteins, hMLH1, has been mapped to chromosome 3 while hMLH2 has been mapped to chromosome 2 and hMLH3 has been mapped to chromosome 7. The polynucleotide sequences of the DNA repair proteins may be used for diagnosis of a hereditary susceptibility to cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:95941 USPATFULL

TITLE: Human DNA mismatch repair proteins

INVENTOR(S): Adams, Mark D., North Potomac, MD, United States

Fleischmann, Robert D., Gaithersburg, MD, United States

Fraser, Claire M., Potomac, MD, United States

Fuldner, Rebecca A., Barnesville, MD, United States

Kirkness, Ewen F., Olney, MD, United States



**Haseltine, William A.**, Washington, DC, United States  
Rosen, Craig A., Laytonsville, MD, United States  
Ruben, Steve, Olney, MD, United States  
Wei, Ying-Fei, Darnestown, MD, United States  
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6380369	B1	20020430
APPLICATION INFO.:	US 1994-294312		19940823 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1994-210143, filed on 16 Mar 1994 Continuation-in-part of Ser. No. US 1994-187757, filed on 27 Jan 1994		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Campbell, Eggerton A.		
LEGAL REPRESENTATIVE:	Human Genome Sciences, Inc.		
NUMBER OF CLAIMS:	62		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	28 Drawing Figure(s); 26 Drawing Page(s)		
LINE COUNT:	1500		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.			

L2 ANSWER 11 OF 78 USPATFULL on STN

TI Method of intracellular binding target molecules

AB The present invention relates to a method by which one can target an undesired target molecule or target antigen, preferably a protein. The method comprises the intracellular expression of an antibody capable of binding to the target. A DNA sequence is delivered to a cell, the DNA sequence contains a sufficient number of nucleotides coding for the portion of an antibody capable of binding to the target operably linked to a promoter that will permit expression of the antibody in the cell(s) of interest. The antibody is then expressed intracellularly and binds to the target, thereby disrupting the target from its normal actions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2001:226439 USPATFULL  
TITLE: Method of intracellular binding target molecules  
INVENTOR(S): Marasco, Wayne A., Wellesley, MA, United States  
**Haseltine, William A.**, Cambridge, MA, United States  
PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Inc., Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6329173	B1	20011211
APPLICATION INFO.:	US 2000-556111		20000421 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1999-287145, filed on 6 Apr 1999, now patented, Pat. No. US 6072036 Division of Ser. No. US 1995-438190, filed on 9 May 1995, now patented, Pat. No. US 5965371 Continuation of Ser. No. US 1993-45274, filed on 31 Mar 1993, now abandoned Continuation-in-part of Ser. No. US 1992-916939, filed on 17 Jul 1992, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Stucker, Jeffrey		
LEGAL REPRESENTATIVE:	Nixon Peabody LLP		
NUMBER OF CLAIMS:	22		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	31 Drawing Figure(s); 17 Drawing Page(s)		

LINE COUNT: 2470  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 12 OF 78 USPATFULL on STN  
TI Human DNA ligase III  
AB A human DNA Ligase III polypeptide and DNA (RNA) encoding such polypeptide and a procedure for producing such polypeptide by recombinant techniques is disclosed. Also disclosed are methods for utilizing such polypeptide via gene therapy for the treatment of disorders associated with a defect in DNA Ligase III. Antagonists against such polypeptides and their use as a therapeutic to destroy unwanted cells are also disclosed. Diagnostic assays to detect mutant DNA Ligase III genes are also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2001:205585 USPATFULL  
TITLE: Human DNA ligase III  
INVENTOR(S): Wei, Ying-Fei, Berkeley, CA, United States  
Yu, Guo-Liang, Berkeley, CA, United States  
Haseltine, William A., Washington, DC, United States  
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., Rockville, MD, 20850  
(non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001041350	A1	20011115
APPLICATION INFO.:	US 2001-879228	A1	20010613 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1998-54775, filed on 3 Apr 1998, GRANTED, Pat. No. US 6284504 Division of Ser. No. US 1995-464402, filed on 5 Jun 1995, GRANTED, Pat. No. US 5858705 Continuation-in-part of Ser. No. WO 1995-US3939, filed on 31 Mar 1995, UNKNOWN		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850		
NUMBER OF CLAIMS:	49		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	10 Drawing Page(s)		
LINE COUNT:	1904		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 13 OF 78 USPATFULL on STN  
TI Human DNA ligase III  
AB A human DNA Ligase III polypeptide and DNA (RNA) encoding such polypeptide and a procedure for producing such polypeptide by recombinant techniques is disclosed. Also disclosed are methods for utilizing such polypeptide via gene therapy for the treatment of disorders associated with a defect in DNA Ligase III. Antagonists against such polypeptides and their use as a therapeutic to destroy unwanted cells are also disclosed. Diagnostic assays to detect mutant DNA Ligase III genes are also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2001:147715 USPATFULL  
TITLE: Human DNA ligase III  
INVENTOR(S): Wei, Ying-Fei, Darnestown, MD, United States  
Yu, Guo-Liang, Darnestown, MD, United States  
Haseltine, William A., NW. Washington, DC, United States  
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
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PATENT INFORMATION:	US 6284504	B1	20010904
APPLICATION INFO.:	US 1998-54775		19980403 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1995-464402, filed on 5 Jun 1995, now patented, Pat. No. US 5858705 Continuation-in-part of Ser. No. WO 1995-US3939, filed on 31 Mar 1995		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Achutamurthy, Ponnathapu		
ASSISTANT EXAMINER:	Tung, Peter P.		
LEGAL REPRESENTATIVE:	Human Genome Sciences Inc.		
NUMBER OF CLAIMS:	22		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	10 Drawing Figure(s); 10 Drawing Page(s)		
LINE COUNT:	1458		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.			

L2 ANSWER 14 OF 78 USPATFULL on STN

TI Method of intracellular binding of target molecules

AB The present invention relates to a method by which one can target an undesired target molecule or target antigen, preferably a protein. The method comprises the intracellular expression of an antibody capable of binding to the target. A DNA sequence is delivered to a cell, the DNA sequence contains a sufficient number of nucleotides coding for the portion of an antibody capable of binding to the target operably linked to a promoter that will permit expression of the antibody in the cell(s) of interest. The antibody is then expressed intracellularly and binds to the target, thereby disrupting the target from its normal actions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2000:70964 USPATFULL  
 TITLE: Method of intracellular binding of target molecules  
 INVENTOR(S): Marasco, Wayne A., Wellesley, MA, United States  
                   Haseltine, William A., Cambridge, MA, United States  
 PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Inc., Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
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PATENT INFORMATION:	US 6072036		20000606
APPLICATION INFO.:	US 1999-287145		19990406 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1995-438190, filed on 9 May 1995, now patented, Pat. No. US 5965371 which is a continuation of Ser. No. US 1993-45274, filed on 31 Mar 1993 which is a continuation-in-part of Ser. No. US 1992-916939, filed on 17 Jul 1992		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Stucker, Jeffrey		
LEGAL REPRESENTATIVE:	Eisenstein, Ronald I., Resnick, David S. Nixon Peabody LLP		
NUMBER OF CLAIMS:	5		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	19 Drawing Figure(s); 17 Drawing Page(s)		
LINE COUNT:	2773		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.			

L2 ANSWER 15 OF 78 USPATFULL on STN

TI Vector comprising a replication competent HIV-1 provirus and a heterologous gene

AB A vector comprising an HIV segment and a heterologous gene segment,

which produces a replication competent and an infective HIV virus is disclosed. When the heterologous gene is a marker gene, the spread of the virus can be observed in both in vitro and in vivo systems. The use of this vector in establishing methods for screening anti-viral compounds is also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2000:27798 USPATFULL  
 TITLE: Vector comprising a replication competent HIV-1 provirus and a heterologous gene  
 INVENTOR(S): **Haseltine, William A.**, Cambridge, MA, United States  
 Terwilliger, Ernest, Boston, MA, United States  
 PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6033902		20000307
APPLICATION INFO.:	US 1992-987572		19921208 (7)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1988-249918, filed on 27 Sep 1988, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Railey, II, Johnny F.		
LEGAL REPRESENTATIVE:	Eisenstein, Ronald I., Resnick, David S., Peabody LLP, Nixon		
NUMBER OF CLAIMS:	11		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	9 Drawing Figure(s); 7 Drawing Page(s)		
LINE COUNT:	737		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 16 OF 78 USPATFULL on STN

TI Vectors containing HIV packaging sequences, packaging defective HIV vectors, and uses thereof  
 AB Packaging defective and packaging proficient HIV vectors are disclosed. These vectors can be used to establish HIV packaging defective cell lines, and to package desired genes. These cell lines can be used in developing a vaccine, HIV antibodies and as part of a system for gene transfer. The packaging proficient vector can be used to target HIV target cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 1999:141683 USPATFULL  
 TITLE: Vectors containing HIV packaging sequences, packaging defective HIV vectors, and uses thereof  
 INVENTOR(S): Sodroski, Joseph G., Medford, MA, United States  
**Haseltine, William A.**, Cambridge, MA, United States  
 Poznansky, Mark, Cambridge, MA, United States  
 Lever, Andrew, Pinner, United Kingdom  
 PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5981276		19991109
APPLICATION INFO.:	US 1997-915429		19970820 (8)
RELATED APPLN. INFO.:	Division of Ser. No. US 1993-152902, filed on 15 Nov 1993, now patented, Pat. No. US 5665577 which is a continuation of Ser. No. US 1992-827588, filed on 29 Jan 1992, now abandoned which is a continuation of Ser. No. US 1990-540746, filed on 20 Jun 1990, now abandoned		

DOCUMENT TYPE: Utility  
FILE SEGMENT: Granted  
PRIMARY EXAMINER: Railey, II, Johnny F.  
LEGAL REPRESENTATIVE: Eisenstein, Ronald I., Resnick, David S.  
NUMBER OF CLAIMS: 11  
EXEMPLARY CLAIM: 1  
NUMBER OF DRAWINGS: 14 Drawing Figure(s); 7 Drawing Page(s)  
LINE COUNT: 1009  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 17 OF 78 USPATFULL on STN

TI Method of intracellular binding of target molecules

AB The present invention relates to a method by which one can target an undesired target molecule or target antigen, preferably a protein. The method comprises the intracellular expression of an antibody capable of binding to the target. A DNA sequence is delivered to a cell, the DNA sequence contains a sufficient number of nucleotides coding for the portion of an antibody capable of binding to the target operably linked to a promoter that will permit expression of the antibody in the cell(s) of interest. The antibody is then expressed intracellularly and binds to the target, thereby disrupting the target from its normal actions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 1999:124707 USPATFULL  
TITLE: Method of intracellular binding of target molecules  
INVENTOR(S): Marasco, Wayne A., Wellesley, MA, United States  
Haseltine, William A., Cambridge, MA, United States  
PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Boston, MA, United States  
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5965371		19991012
APPLICATION INFO.:	US 1995-438190		19950509 (8)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1993-45274, filed on 31 Mar 1993 which is a continuation-in-part of Ser. No. US 1992-916939, filed on 17 Jul 1992, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Stucker, Jeffrey		
LEGAL REPRESENTATIVE:	Eisenstein, Ronald I., Conlin, David G., Resnick, David S.		
NUMBER OF CLAIMS:	101		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	33 Drawing Figure(s); 17 Drawing Page(s)		
LINE COUNT:	3086		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 18 OF 78 USPATFULL on STN

TI Polynucleotides encoding human DNA ligase III and methods of using these polynucleotides

AB A human DNA Ligase III polypeptide and DNA (RNA) encoding such polypeptide and a procedure for producing such polypeptide by recombinant techniques are disclosed. Also disclosed are methods for utilizing such polypeptide via gene therapy for the treatment of disorders associated with a defect in DNA Ligase III. Antagonists against such polypeptides and their use as a therapeutic to destroy unwanted cells are also disclosed. Diagnostic assays to detect mutant DNA Ligase III genes are also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 1999:4370 USPATFULL  
TITLE: Polynucleotides encoding human DNA ligase III and

INVENTOR(S): methods of using these polynucleotides  
 Wei, Ying-Fei, Darnestown, MD, United States  
 Yu, Guo-Liang, Darnestown, MD, United States  
**Haseltine, William A.**, Washington, DC, United States  
 PATENT ASSIGNEE(S): Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5858705		19990112
APPLICATION INFO.:	US 1995-464402		19950605 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Walsh, Stephen		
ASSISTANT EXAMINER:	Lathrop, Brian		
LEGAL REPRESENTATIVE:	Olstein, Elliot M., Mullins, J. G.		
NUMBER OF CLAIMS:	19		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	12 Drawing Figure(s); 12 Drawing Page(s)		
LINE COUNT:	1615		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 19 OF 78 USPATFULL on STN

TI Immunogenic peptides, antibodies and uses thereof relating to CD4 receptor binding  
 AB Immunogenic peptides containing amino acid residues which define a binding site to a CD4 receptor are disclosed. Antibodies to these peptides are also disclosed. Methods of reducing the ability of a gp120 env protein to bind to CD4 are also disclosed. Methods of treatment and prophylaxis using these antibodies and peptides are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 1999:4039 USPATFULL  
 TITLE: Immunogenic peptides, antibodies and uses thereof relating to CD4 receptor binding  
 INVENTOR(S): Sodroski, Joseph G., Medford, MA, United States  
**Haseltine, William A.**, Boston, MA, United States  
 Olshevsky, Udy, Remath-OAN, Israel  
 Helseth, Eirik, Trondheim, Norway  
 Furman, Craig D., Nashua, NH, United States  
 PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5858366		19990112
APPLICATION INFO.:	US 1993-135312		19931012 (8)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1991-669072, filed on 14 Mar 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-524632, filed on 16 May 1990, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Smith, Lynette F.		
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I. Dike, Bronstein, Roberts & Cushman, LLP		
NUMBER OF CLAIMS:	8		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	8 Drawing Figure(s); 8 Drawing Page(s)		
LINE COUNT:	1226		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 20 OF 78 USPATFULL on STN

TI Reactive neutralizing human anti-GP120 recombinant antibody, DNA coding the same and use thereof

AB The present invention is directed to a recombinant human monoclonal antibody which binds to a discontinuous epitope on the HIV gp120 envelope glycoprotein, blocks the binding of gp120 to the CD4 receptor, and neutralizes a broad range of HIV isolates. The present invention also provides the primary nucleotide and deduced amino acid sequences of the rearranged heavy and light chains of the recombinant monoclonal antibody of the present invention, and a method of screening for antibodies which block binding of envelope glycoprotein to the CD4 receptor.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 1998:160114 USPATFULL

TITLE: Reactive neutralizing human anti-GP120 recombinant antibody, DNA coding the same and use thereof

INVENTOR(S): Sodroski, Joseph G., Medford, MA, United States  
Marasco, Wayne A., Wellesley, MA, United States  
Posner, Marshall R., Dedham, MA, United States  
**Haseltine, William A.**, Cambridge, MA, United States

PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Boston, MA, United States (U.S. corporation)  
New England Deaconess Hospital Corp., Dedham, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5852186		19981222
APPLICATION INFO.:	US 1995-480774		19950607 (8)
RELATED APPLN. INFO.:	Division of Ser. No. US 1995-400674, filed on 8 Mar 1995, now abandoned which is a continuation of Ser. No. US 1991-804652, filed on 10 Dec 1991, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Budens, Robert D.		
LEGAL REPRESENTATIVE:	Conlin, David G., Resnick, David S., Eisenstein, Ronald I.		
NUMBER OF CLAIMS:	6		
EXEMPLARY CLAIM:	1,2		
NUMBER OF DRAWINGS:	22 Drawing Figure(s); 16 Drawing Page(s)		
LINE COUNT:	2191		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 21 OF 78 USPATFULL on STN

TI Method of intracellular binding of target molecules

AB The present invention relates to a method by which one can target an undesired target molecule or target antigen, preferably a protein. The method comprises the intracellular expression of an antibody capable of binding to the target. A DNA sequence is delivered to a cell, the DNA sequence contains a sufficient number of nucleotides coding for the portion of an antibody capable of binding to the target operably linked to a promoter that will permit expression of the antibody in the cell(s) of interest. The antibody is then expressed intracellularly and binds to the target, thereby disrupting the target from its normal actions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 1998:159761 USPATFULL

TITLE: Method of intracellular binding of target molecules

INVENTOR(S): Marasco, Wayne A., Wellesley, MA, United States  
**Haseltine, William A.**, Rockville, MD, United States

PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Boston, MA, United States

(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5851829		19981222
	WO 9402610		19940203
APPLICATION INFO.:	US 1995-373190		19950330 (8)
	WO 1993-US6735		19930716
			19950330 PCT 371 date
			19950330 PCT 102(e) date
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Stucker, Jeffrey		
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I., Resnick, David S.		
NUMBER OF CLAIMS:	62		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	34 Drawing Figure(s); 19 Drawing Page(s)		
LINE COUNT:	3209		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 22 OF 78 USPATFULL on STN

TI Immunogenic peptides, antibodies and uses thereof relating to CD4 receptor binding

AB Immunogenic peptides containing amino acid residues which define a binding site to a CD4 receptor are disclosed. Antibodies to these peptides are also disclosed. Methods of reducing the ability of a gp120 env protein to bind to CD4 are also disclosed. Methods of treatment and prophylaxis using these antibodies and peptides are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 1998:122077 USPATFULL

TITLE: Immunogenic peptides, antibodies and uses thereof relating to CD4 receptor binding

INVENTOR(S): Sodroski, Joseph G., Medford, MA, United States  
**Haseltine, William A.**, Canbridge, MA, United States  
Furman, Craig D., Nashua, NH, United States  
Olshevsky, Udy, Remath-Oan, Israel  
Helseth, Eirik, Trondheim, Norway  
Wyatt, Richard, Tewksbury, MA, United States  
Thali, Markus, Brookline, MA, United States

PATENT ASSIGNEE(S): Dana-Farber Cancer Instistute, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5817316		19981006
APPLICATION INFO.:	US 1992-858165		19920326 (7)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1991-669072, filed on 14 Mar 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-524632, filed on 16 May 1990, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Smith, Lynette F.		
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I. Dike, Bronstein, Roberts & Cushman		
NUMBER OF CLAIMS:	11		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	8 Drawing Figure(s); 8 Drawing Page(s)		
LINE COUNT:	1354		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.



L2 ANSWER 23 OF 78 USPATFULL on STN

TI Assays for factors affecting circularization of DNA, assays for factors affecting DNA integration, factors, and uses thereof

AB An assay for factors that affect integration of DNA into target DNA is disclosed. Assays for methods of screening for factors which effect viral DNA circularization either by homologous recombination, end-to-end ligation, or autointegration, are also disclosed. A method for screening for factors which will enhance circularization rather than integration by testing cellular cytoplasmic fluid under conditions which permit circularization in the fluid is also described. Factors which effect integration and circularization are disclosed. Therapeutic methods for retarding viral infection are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 1998:61388 USPATFULL

TITLE: Assays for factors affecting circularization of DNA, assays for factors affecting DNA integration, factors, and uses thereof

INVENTOR(S): **Haseltine, William A.**, Cambridge, MA, United States

Farnet, Christopher M., Cambridge, MA, United States  
PATENT ASSIGNEE(S): Dana Farber Cancer Institute, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5759768		19980602
APPLICATION INFO.:	US 1995-425726		19950420 (8)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1991-703180, filed on 17 May 1991, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Degen, Nancy		
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I. Dike, Bronstein, Roberts & Cushman, LLP		
NUMBER OF CLAIMS:	27		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	40 Drawing Figure(s); 25 Drawing Page(s)		
LINE COUNT:	1672		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 24 OF 78 USPATFULL on STN

TI Vectors containing HIV packaging sequences, packaging defective HIV vectors, and uses thereof

AB Packaging defective and packaging proficient HIV vectors are disclosed. These vectors can be used to establish HIV packaging defective cell lines, and to package desired genes. These cell lines can be used in developing a vaccine, HIV antibodies and as part of a system for gene transfer. The packaging proficient vector can be used to target HIV target cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 97:81129 USPATFULL

TITLE: Vectors containing HIV packaging sequences, packaging defective HIV vectors, and uses thereof

INVENTOR(S): Sodroski, Joseph G., Medford, MA, United States

**Haseltine, William A.**, Cambridge, MA, United States

Poznansky, Mark, Cambridge, MA, United States

Lever, Andrew, Pinner Middlesex, England

Gottlinger, Heinrich, Munich, Germany, Federal Republic of

PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
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PATENT INFORMATION:	US 5665577		19970909
APPLICATION INFO.:	US 1993-152902		19931115 (8)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1992-827588, filed on 29 Jan 1992, now abandoned which is a continuation-in-part of Ser. No. US 1990-540746, filed on 20 Jun 1990, now abandoned which is a continuation-in-part of Ser. No. US 1989-307664, filed on 6 Feb 1989, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Fleisher, Mindy		
ASSISTANT EXAMINER:	Railey, II, Johnny F.		
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I.		
NUMBER OF CLAIMS:	72		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	8 Drawing Figure(s); 8 Drawing Page(s)		
LINE COUNT:	1156		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.			

L2 ANSWER 25 OF 78 USPATFULL on STN

TI Vectors expressing hybrid viruses, methods of use and novel assays

AB A vector which can be used to establish a hybrid SIV/HIV-1 virus is described. This virus can be used to infect an animal such as a monkey to establish an animal model for in vivo testing. This animal model can be used for purposes such as screening for therapeutics, adjuvants and vaccines.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 97:68366 USPATFULL

TITLE: Vectors expressing hybrid viruses, methods of use and novel assays

INVENTOR(S): Sodroski, Joseph, Medford, MA, United States  
**Haseltine, William A.**, Cambridge, MA, United States  
 Letvin, Norman, Newton, MA, United States  
 Li, John, Boston, MA, United States

PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
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PATENT INFORMATION:	US 5654195		19970805
APPLICATION INFO.:	US 1994-268799		19940701 (8)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1992-887505, filed on 22 May 1992, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Guzo, David		
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I.		
NUMBER OF CLAIMS:	19		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	8 Drawing Figure(s); 6 Drawing Page(s)		
LINE COUNT:	1388		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.			

L2 ANSWER 26 OF 78 USPATFULL on STN

TI YC1 gene

AB Isolated and purified YC1 genes and proteins are disclosed. The protein binds to a site in the HIV-LTR, the NRE-1 site, and can inhibit the expression of a gene operably linked to the HIV-1 LTR. The use of the protein and gene are discussed. Repressible and inducible expression systems using the YC1 gene are also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 97:66033 USPATFULL  
TITLE: YC1 gene  
INVENTOR(S): Lu, Yinchun, Wellesley, MA, United States  
Haseltine, William A., Cambridge, MA, United States  
PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Boston, MA, United States  
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5652144		19970729
APPLICATION INFO.:	US 1992-973431		19921110 (7)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Guzo, David		
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I.		
NUMBER OF CLAIMS:	4		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	12 Drawing Figure(s); 8 Drawing Page(s)		
LINE COUNT:	1256		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 27 OF 78 USPATFULL on STN

TI Cis-acting repression sequences, cis-acting antirepression sequences, vectors, methods of preparation and use

AB Cis-acting repression sequences which are able to provide a cis-acting inhibitory effect on the expression of a gene when placed downstream of the gene in its untranslated message are disclosed. Cis-acting anti-repression sequences which can relieve the cis-acting repression in the presence of the art gene product are also disclosed. These sequences correspond to a sufficient number of nucleotides from the HIV-I, HIV-2, STLV-3 or HTLV-IV genomes to provide the repression or anti-repression effects. The use of the sequences in vectors and systems to control the expression of a desired gene product is also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 97:14591 USPATFULL  
TITLE: Cis-acting repression sequences, cis-acting antirepression sequences, vectors, methods of preparation and use  
INVENTOR(S): Haseltine, William A., Cambridge, MA, United States  
Rosen, Craig A., Glen Ridge, NJ, United States  
Sodroski, Joseph G., Cambridge, MA, United States  
Terwilliger, Ernest, Boston, MA, United States  
Goh, Wei C., Stanford, CA, United States  
PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Boston, MA, United States  
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5604114		19970218
APPLICATION INFO.:	US 1993-41887		19930402 (8)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1992-847854, filed on 9 Mar 1992, now abandoned which is a continuation of Ser. No. US 1990-591667, filed on 27 Sep 1990, now abandoned which is a continuation of Ser. No. US 1987-56620, filed on 29 May 1987, now abandoned which is a continuation-in-part of Ser. No. US 1986-865151, filed on 20 May 1986, now patented, Pat. No. US 4935372		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		

PRIMARY EXAMINER: Elliott, George C.  
ASSISTANT EXAMINER: Railey, II, Johnny F.  
LEGAL REPRESENTATIVE: Conlin, David G., Eisenstein, Ronald I.  
NUMBER OF CLAIMS: 27  
EXEMPLARY CLAIM: 1  
NUMBER OF DRAWINGS: 20 Drawing Figure(s); 11 Drawing Page(s)  
LINE COUNT: 1434  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 28 OF 78 USPATFULL on STN  
TI H. saimiri-HTLV-X region vector  
AB An H. saimiri-HTLV-1 or 2 X region vector is disclosed. This vector can be used to establish continuous cell lines of difficult to grow cells, such as human T-cells. It can also be used to obtain certain cell products and in methods for screening new compounds.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 95:52244 USPATFULL  
TITLE: H. saimiri-HTLV-X region vector  
INVENTOR(S): **Haseltine, William A.**, Cambridge, MA, United States  
McGuire, Kathleen, Jamaica Plain, MA, United States  
Dokhelar, Marie-Christine, Paris, France  
Grassmann, Ralph, Erlangen, Germany, Federal Republic of  
Fleckenstein, Bernard, Weisenthau, Germany, Federal Republic of  
Muller-Fleckenstein, Ingrid, Weisenthau, Germany, Federal Republic of  
PATENT ASSIGNEE(S): Dana Farber Cancer Institute, Boston, MA, United States (U.S. corporation)  
Behringwerke Aktiengesellschaft, Frankfurt, Germany, Federal Republic of (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5424197		19950613
APPLICATION INFO.:	US 1992-976661		19921116 (7)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1992-816774, filed on 2 Jan 1992, now abandoned which is a continuation of Ser. No. US 1988-254416, filed on 6 Oct 1988, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Stone, Jacqueline		
ASSISTANT EXAMINER:	Railey, II, Johnny F.		
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I.		
NUMBER OF CLAIMS:	16		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	11 Drawing Figure(s); 6 Drawing Page(s)		
LINE COUNT:	777		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 29 OF 78 USPATFULL on STN  
TI Art (rev) protein of human T-cell leukemia virus  
AB A gene and gene product that regulates the expression of the capsid envelope genes of HTLV-III/LAV and that can be used to regulate the expression of heterologous (non-viral) genes as well is disclosed. This art gene consists of two exons and can be used in creating nucleotide segments, vectors and cell lines. A new method for screening for compounds that inhibit the replication of HTLV-III is also described and comprises:

(1) transfecting a T-cell line with the HTLV-III art and env genes;

(2) thereafter, adding a preselected compound to the transformed cell line in increasing concentrations; and

(3) determining whether the compound effects the art function without being toxic to the cell.

An additional parameter to use in diagnosis of AIDS disease is also described. The use of the art gene and gene product in AIDS therapy is also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 94:51511 USPATFULL  
TITLE: Art (rev) protein of human T-cell leukemia virus  
INVENTOR(S): **Haseltine, William A.**, Cambridge, MA, United States  
Rosen, Craig A., Brookline, MA, United States  
Sodroski, Joseph G., Cambridge, MA, United States  
Goh, Wei C., Somerville, MA, United States  
PATENT ASSIGNEE(S): Dana Farber Cancer Institute, Boston, MA, United States  
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5321124		19940614
APPLICATION INFO.:	US 1992-995948		19921218 (7)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1990-538189, filed on 14 Jun 1990, now abandoned which is a division of Ser. No. US 1986-865151, filed on 20 May 1986, now patented, Pat. No. US 4935372		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Low, Christopher S. F.		
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I.		
NUMBER OF CLAIMS:	4		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	17 Drawing Figure(s); 11 Drawing Page(s)		
LINE COUNT:	1062		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 30 OF 78 USPATFULL on STN

TI Sequences containing the vpu gene and vectors therefore methods of preparation and use

AB DNA segments encoding the vpu gene and a vector encoding the vpu gene are disclosed. These sequences containing the vpu gene can be used to express a protein that has antigenic determinants that can be used to screen for people having the HIV-1 virus.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 94:15664 USPATFULL  
TITLE: Sequences containing the vpu gene and vectors therefore methods of preparation and use  
INVENTOR(S): **Haseltine, William A.**, Cambridge, MA, United States  
Terwilliger, Ernest, Boston, MA, United States  
Cohen, Eric, Brighton, MA, United States  
PATENT ASSIGNEE(S): Dana Farber Cancer Institute, Boston, MA, United States  
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5288640		19940222
APPLICATION INFO.:	US 1991-716131		19910617 (7)
RELATED APPLN. INFO.:	Division of Ser. No. US 1988-193321, filed on 12 May 1988, now patented, Pat. No. US 5043262		

DOCUMENT TYPE: Utility  
FILE SEGMENT: Granted  
PRIMARY EXAMINER: Nucker, Christine M.  
ASSISTANT EXAMINER: Barnd, D. L.  
LEGAL REPRESENTATIVE: Conlin, David G., Eisenstein, Ronald I.  
NUMBER OF CLAIMS: 5  
EXEMPLARY CLAIM: 1  
NUMBER OF DRAWINGS: 14 Drawing Figure(s); 8 Drawing Page(s)  
LINE COUNT: 493  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 31 OF 78 USPATFULL on STN

TI Expression of human immunodeficiency virus (HIV) reverse transcriptase  
AB This invention describes pHRT25, a plasmid containing a modified pol gene of the Human Immunodeficiency Virus Type 1 (HIV-1), formerly HTLV-III, under control of an inducible trp promoter. Methods of expressing reverse transcriptase activity using pHRT25 in E. coli are described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 93:89562 USPATFULL  
TITLE: Expression of human immunodeficiency virus (HIV) reverse transcriptase  
INVENTOR(S): Goff, Stephen P., Tenaflly, NJ, United States  
Tanese, Naoko, New York, NY, United States  
**Haseltine, William A.**, Cambridge, MA, United States  
PATENT ASSIGNEE(S): The Trustees of Columbia University in the City of New York, New York, NY, United States (U.S. corporation)  
The Dana Farber Cancer Institute, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5256554		19931026
APPLICATION INFO.:	US 1991-800682		19911202 (7)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1990-552848, filed on 12 Jul 1990, now abandoned which is a continuation of Ser. No. US 1986-865156, filed on 20 May 1986, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Schwartz, Richard A.		
ASSISTANT EXAMINER:	Railey II, Johnny F.		
LEGAL REPRESENTATIVE:	White, John P.		
NUMBER OF CLAIMS:	1		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	27 Drawing Figure(s); 26 Drawing Page(s)		
LINE COUNT:	462		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 32 OF 78 USPATFULL on STN

TI Gene expressing VPT protein and vectors expressing this protein  
AB Viral protein T from Human Immunodeficiency Virus Type 1 (HIV-1) is disclosed. The protein has a molecular weight of approximately 17 kD and is produced by the vpt gene of HIV-1. This protein is antigenic. Vectors capable of expressing the vpt protein are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 93:31326 USPATFULL  
TITLE: Gene expressing VPT protein and vectors expressing this protein  
INVENTOR(S): **Haseltine, William A.**, Cambridge, MA, United States  
Cohen, Eric, Brighton, MA, United States

PATENT ASSIGNEE(S): Dana Farber Cancer Institute, Boston, MA, United States  
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5204258		19930420
APPLICATION INFO.:	US 1989-360847		19890602 (7)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Schwartz, Richard A.		
ASSISTANT EXAMINER:	Railey, II, Johnny F.		
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I.		
NUMBER OF CLAIMS:	7		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	10 Drawing Figure(s); 8 Drawing Page(s)		
LINE COUNT:	473		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 33 OF 78 USPATFULL on STN

TI Protein, sequences containing the VPU gene therefore, vectors, methods of preparation and use

AB A protein having molecular weight of approximately 16 kD which is also cleaved into a protein having a molecular weight of 15 kD is disclosed. This protein is referred to as viral protein U and produced by the vpu gene. It is disclosed that this protein has antigenic determinants and can be used to screen for people having the HIV-1 virus.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 91:68814 USPATFULL

TITLE: Protein, sequences containing the VPU gene therefore, vectors, methods of preparation and use

INVENTOR(S): **Haseltine, William A.**, Cambridge, MA, United States  
Terwilliger, Ernest, Boston, MA, United States  
Cohen, Eric, Brighton, MA, United States

PATENT ASSIGNEE(S): Dana Farber Cancer Institute, Boston, MA, United States  
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5043262		19910827
APPLICATION INFO.:	US 1988-193321		19880512 (7)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Schain, Howard E.		
ASSISTANT EXAMINER:	Baker, K. Keith		
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I.		
NUMBER OF CLAIMS:	15		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	8 Drawing Figure(s); 8 Drawing Page(s)		
LINE COUNT:	520		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 34 OF 78 USPATFULL on STN

TI Stable TatIII cell lines, TatIII gene products, and assay methods

AB This invention describes stable tat.sub.III cell lines. It is disclosed that by transfecting a preselected tat.sub.III cell line with a vector containing a sufficient amount of the HTLV-III LTR responsive to tat.sub.III gene products for trans-activation and an enhancer upstream of the tat.sub.III responsive segment, it is possible to express high levels of the tat.sub.III gene products. By including a preselected heterologous gene on this vector, it is also possible to express high levels of a desired gene product. A substantially pure protein comprising 86 amino acids and having an apparent molecular weight of

about 14,000 dalton and exhibiting trans-activating activity is also disclosed. This protein and polypeptides having trans-activating ability, which is also disclosed, can be used to produce high levels of a desired gene product. A method of detecting the presence of HTLV-III/LAV virus in an individual is also disclosed and comprises the step of:

(a) incubating whole blood or lymphocytes from the individual to be tested with tat.sub.III cell lines of the present invention in a culture medium; and

(b) screening for cytopathic effects on the cells is also disclosed. A method of screening for a compound that inhibits trans-activation of the tat.sub.III gene product is also disclosed and comprises the steps of:

(1) transfecting a tat.sub.III cell line of the present invention with a vector containing a gene that expresses a selectable marker and whose expression is under the control of an HTLV-III LTR;

(2) transfecting the same type of tat.sub.III cell lines as in step (1) with the selectable gene chosen in step (1) but under the control of a different regulatory sequence;

(3) thereafter, adding a preselected compound to each of the cell lines in increasing concentrations; and

(4) measuring the expression of the selectable gene product to determine whether the compound effects the tat.sub.III function without being toxic to the cell.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 91:1087 USPATFULL  
 TITLE: Stable TatIII cell lines, TatIII gene products, and assay methods  
 INVENTOR(S): Haseltine, William A., Cambridge, MA, United States  
 Rosen, Craig A., Brookline, MA, United States  
 Sodroski, Joseph G., Cambridge, MA, United States  
 Goh, Wei C., Somerville, MA, United States  
 PATENT ASSIGNEE(S): Dana Farber Cancer Institute, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 4981790		19910101
APPLICATION INFO.:	US 1985-806263		19851206 (6)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1984-614297, filed on 25 May 1984		

	NUMBER	DATE
PRIORITY INFORMATION:	CA 1985-482374	19850524
	WO 1985-US985	19850524
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	Granted	
PRIMARY EXAMINER:	Teskin, Robin L.	
ASSISTANT EXAMINER:	Burrous, Beth A.	
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I.	
NUMBER OF CLAIMS:	16	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	14 Drawing Figure(s); 7 Drawing Page(s)	
LINE COUNT:	847	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.



L2 ANSWER 35 OF 78 USPATFULL on STN  
TI Peptides for the diagnosis of HTLV-III antibodies, their preparation and use  
AB Certain peptide fragments of the human T-cell leukemia (lymphotropic) virus (HTLV-III) are particularly immunoreactive to HTLV-III antibodies, and can therefore be applied to immunodiagnostic tests for the detection of antibodies to HTLV-III.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 88:40574 USPATFULL  
TITLE: Peptides for the diagnosis of HTLV-III antibodies, their preparation and use  
INVENTOR(S): Beltz, Gerald A., Lexington, MA, United States  
Thorn, Richard M., Milford, MA, United States  
Marciani, Dante J., Hopkinton, MA, United States  
Hung, Chung-Ho, Milford, MA, United States  
Haseltine, William A., Cambridge, MA, United States  
PATENT ASSIGNEE(S): Cambridge Bioscience Corporation, Hopkinton, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 4753873		19880628
APPLICATION INFO.:	US 1986-825597		19860203 (6)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1985-819917, filed on 6 Nov 1985		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Nucker, Christine M.		
LEGAL REPRESENTATIVE:	Saidman, Sterne, Kessler & Goldstein		
NUMBER OF CLAIMS:	17		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	21 Drawing Figure(s); 22 Drawing Page(s)		
LINE COUNT:	1122		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 36 OF 78 USPATFULL on STN  
TI Trans-acting transcriptional factors  
AB This invention describes the discovery of a novel phenomena in retrovirus transcription, namely transcriptional trans-activation. Described herein are novel trans-acting factors which may be employed to enhance the production of heterologous genes. Described is a novel trans-acting directing gene, designated herein as the "luk" gene and the 35,000 to 45,000, more specifically about 42,000 dalton molecular weight protein encoded thereby.

The present invention demonstrates the LTR elements of HTLV can function as transcriptional promoters for heterologous genes on both unintegrated and integrated DNA. In general, the HTLV-1 LTR is a stronger promoter than is the HTLV-II LTR in its requirements for cellular and/or viral trans-acting factors in order to function efficiently. HTLV infection results in the production of trans-acting factors that dramatically increase the rate of HTLV LTR-promoted transcription.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 88:24366 USPATFULL  
TITLE: Trans-acting transcriptional factors  
INVENTOR(S): Haseltine, William A., Cambridge, MA, United States  
Sodrowski, Joseph G., Cambridge, MA, United States  
Rosen, Craig A., Brookline, MA, United States  
PATENT ASSIGNEE(S): Dana Farber Cancer Institute, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 4738922		19880419
APPLICATION INFO.:	US 1984-614297		19840525 (6)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Wiseman, Thomas G.		
ASSISTANT EXAMINER:	Seidman, S.		
LEGAL REPRESENTATIVE:	Conlin, David G., Linek, Ernest V., Eisenstein, Ronald I.		
NUMBER OF CLAIMS:	26		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	3 Drawing Figure(s); 3 Drawing Page(s)		
LINE COUNT:	767		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 37 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 TI Human DNA mismatch repair protein.  
 AB The present invention discloses three human DNA repair proteins and DNA (RNA) encoding such proteins. The DNA repair proteins may be produced by recombinant DNA techniques. One of the human DNA repair proteins, hmlh1, has been mapped on chromosome 3. The polynucleotide sequences of DNA repair proteins may be used for diagnosis of a hereditary susceptibility to cancer.

ACCESSION NUMBER: 2003:484626 BIOSIS  
 DOCUMENT NUMBER: PREV200300484626  
 TITLE: Human DNA mismatch repair protein.  
 AUTHOR(S): **Haseltine, William A.** [Inventor, Reprint Author]; Ruben, Steven [Inventor]; Wei, Ying-Fei [Inventor]; Adams, Mark D. [Inventor]; Fleischmann, Robert D. [Inventor]; Fraser, Claire M. [Inventor]; Rosen, Craig A. [Inventor]; Fuldner, Rebecca A. [Inventor]; Kirkness, Ewen F. [Inventor]

CORPORATE SOURCE: ASSIGNEE: Human Genome Sciences, Inc.  
 PATENT INFORMATION: US 6620619 September 16, 2003  
 SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Sep 16 2003) Vol. 1274, No. 3.  
<http://www.uspto.gov/web/menu/patdata.html>. e-file.  
 ISSN: 0098-1133 (ISSN print).

DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 15 Oct 2003  
 Last Updated on STN: 15 Oct 2003

L2 ANSWER 38 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 TI Human DNA mismatch repair proteins.  
 AB The present invention discloses three human DNA repair proteins and DNA (RNA) encoding such proteins and a procedure for producing such proteins by recombinant techniques. One of the human DNA repair proteins, hMLH1, has been mapped to chromosome 3 while hMLH2 has been mapped to chromosome 2 and hMLH3 has been mapped to chromosome 7. The polynucleotide sequences of the DNA repair proteins may be used for therapeutic and diagnostic treatments of a hereditary susceptibility to cancer.

ACCESSION NUMBER: 2003:435948 BIOSIS  
 DOCUMENT NUMBER: PREV200300435948  
 TITLE: Human DNA mismatch repair proteins.  
 AUTHOR(S): **Haseltine, William A.** [Inventor, Reprint Author]; Ruben, Steven M. [Inventor]; Wei, Ying-Fei [Inventor]; Adams, Mark D. [Inventor]; Fleischmann, Robert D. [Inventor]; Fraser, Claire M. [Inventor]; Fuldner, Rebecca A. [Inventor]; Kirkness, Ewen F. [Inventor]; Rosen, Craig A. [Inventor]; Vogelstein, Bert [Inventor]; Kinzler, Kenneth W. [Inventor]; Nicolaides, Nicholas C. [Inventor];

CORPORATE SOURCE: Papadopoulos, Nickolas [Inventor]  
Brookeville, MD, USA  
ASSIGNEE: Human Genome Sciences, Inc.; The Johns Hopkins University  
PATENT INFORMATION: US 6610477 August 26, 2003  
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Aug. 26, 2003) Vol. 1273, No. 4.  
<http://www.uspto.gov/web/menu/patdata.html>. e-file.  
ISSN: 0098-1133 (ISSN print).  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
ENTRY DATE: Entered STN: 17 Sep 2003  
Last Updated on STN: 17 Sep 2003

L2 ANSWER 39 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Human DNA mismatch repair polynucleotides.  
AB The present invention discloses three human DNA repair proteins and DNA (RNA) encoding such proteins. The DNA repair proteins may be produced by recombinant DNA techniques. One of the human DNA repair proteins, hmlh1, has been mapped on chromosome 3. The polynucleotide sequences of DNA repair proteins may be used for diagnosis of a hereditary susceptibility to cancer.

ACCESSION NUMBER: 2003:53897 BIOSIS  
DOCUMENT NUMBER: PREV200300053897  
TITLE: Human DNA mismatch repair polynucleotides.  
AUTHOR(S): Adams, Mark D. [Inventor, Reprint Author]; Fleischmann, Robert D. [Inventor]; Fraser, Claire M. [Inventor]; Fuldner, Rebecca A. [Inventor]; Kirkness, Ewen F. [Inventor]; Haseltine, William A. [Inventor]; Rosen, Craig A. [Inventor]; Ruben, Steve [Inventor]; Wei, Ying-Fei [Inventor]  
CORPORATE SOURCE: Queenstown, MD, USA  
ASSIGNEE: Human Genome Sciences, Inc.  
PATENT INFORMATION: US 6482606 November 19, 2002  
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Nov. 19, 2002) Vol. 1264, No. 3.  
<http://www.uspto.gov/web/menu/patdata.html>. e-file.  
ISSN: 0098-1133 (ISSN print).  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
ENTRY DATE: Entered STN: 22 Jan 2003  
Last Updated on STN: 22 Jan 2003

L2 ANSWER 40 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Human DNA Ligase IV.  
AB A human DNA Ligase IV polypeptide and DNA (RNA) encoding such polypeptide and a procedure for producing such polypeptide by recombinant techniques is disclosed. Also disclosed are methods for utilizing such polypeptide via gene therapy for the treatment of disorders associated with a defect in DNA Ligase IV. Antagonists against such polypeptides and their use as a therapeutic to destroy unwanted cells are also disclosed. Diagnostic assays to detect mutant DNA Ligase IV genes are also disclosed.

ACCESSION NUMBER: 2002:584537 BIOSIS  
DOCUMENT NUMBER: PREV200200584537  
TITLE: Human DNA Ligase IV.  
AUTHOR(S): Wei, Ying-Fei [Inventor]; Haseltine, William A. [Inventor]  
CORPORATE SOURCE: Human Genome Sciences, Inc.  
PATENT INFORMATION: US 6455274 September 24, 2002  
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Sep. 24, 2002) Vol. 1262, No. 4.  
<http://www.uspto.gov/web/menu/patdata.html>. e-file.  
CODEN: OGUPE7. ISSN: 0098-1133.  
DOCUMENT TYPE: Patent

LANGUAGE: English  
ENTRY DATE: Entered STN: 13 Nov 2002  
Last Updated on STN: 13 Nov 2002

L2 ANSWER 41 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Human DNA mismatch repair proteins.  
AB The invention discloses three human DNA repair proteins and DNA (RNA) encoding such proteins and a procedure for producing such proteins by recombinant techniques. One of the human DNA repair proteins, hMLH1, has been mapped to chromosome 3 while hMLH2 has been mapped to chromosome 2 and hMLH3 has been mapped to chromosome 7. The polynucleotide sequences of the DNA repair proteins may be used for therapeutic and diagnostic treatments of a hereditary susceptibility to cancer.

ACCESSION NUMBER: 2002:447022 BIOSIS  
DOCUMENT NUMBER: PREV200200447022  
TITLE: Human DNA mismatch repair proteins.  
AUTHOR(S): **Haseltine, William A.** [Inventor, Reprint author];  
Ruben, Steven M. [Inventor]; Wei, Ying-Fei [Inventor];  
Adams, Mark D. [Inventor]; Fleischmann, Robert D.  
[Inventor]; Fraser, Claire M. [Inventor]; Fuldner, Rebecca  
A. [Inventor]; Kirkness, Ewen F. [Inventor]; Rosen, Craig  
A. [Inventor]  
CORPORATE SOURCE: Washington, DC, USA  
ASSIGNEE: Human Genome Sciences, Inc.  
PATENT INFORMATION: US 6416984 July 09, 2002  
SOURCE: Official Gazette of the United States Patent and Trademark  
Office Patents, (July 9, 2002) Vol. 1260, No. 2.  
<http://www.uspto.gov/web/menu/patdata.html>. e-file.  
CODEN: OGUPE7. ISSN: 0098-1133.  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
ENTRY DATE: Entered STN: 21 Aug 2002  
Last Updated on STN: 21 Aug 2002

L2 ANSWER 42 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Human DNA mismatch repair proteins.  
AB The present invention discloses three human DNA repair proteins and DNA (RNA) encoding such proteins. The DNA repair proteins which may be produced by recombinant DNA techniques. One of the human DNA repair proteins, hMLH1, has been mapped to chromosome 3 while hMLH2 has been mapped to chromosome 2 and hMLH3 has been mapped to chromosome 7. The polynucleotide sequences of the DNA repair proteins may be used for diagnosis of a hereditary susceptibility to cancer.

ACCESSION NUMBER: 2002:308881 BIOSIS  
DOCUMENT NUMBER: PREV200200308881  
TITLE: Human DNA mismatch repair proteins.  
AUTHOR(S): Adams, Mark D. [Inventor, Reprint author]; Fleischmann,  
Robert D. [Inventor]; Fraser, Claire M. [Inventor];  
Fuldner, Rebecca A. [Inventor]; Kirkness, Ewen F.  
[Inventor]; **Haseltine, William A.** [Inventor];  
Rosen, Craig A. [Inventor]; Ruben, Steve [Inventor]; Wei,  
Ying-Fei [Inventor]  
CORPORATE SOURCE: North Potomac, MD, USA  
ASSIGNEE: Human Genome Sciences, Inc.  
PATENT INFORMATION: US 6380369 April 30, 2002  
SOURCE: Official Gazette of the United States Patent and Trademark  
Office Patents, (Apr. 30, 2002) Vol. 1257, No. 5.  
<http://www.uspto.gov/web/menu/patdata.html>. e-file.  
CODEN: OGUPE7. ISSN: 0098-1133.  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
ENTRY DATE: Entered STN: 22 May 2002  
Last Updated on STN: 22 May 2002

L2 ANSWER 43 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Method of intracellular binding target molecules.  
AB The present invention relates to a method by which one can target an undesired target molecule or target antigen, preferably a protein. The method comprises the intracellular expression of an antibody capable of binding to the target. A DNA sequence is delivered to a cell, the DNA sequence contains a sufficient number of nucleotides coding for the portion of an antibody capable of binding to the target operably linked to a promoter that will permit expression of the antibody in the cell(s) of interest. The antibody is then expressed intracellularly and binds to the target, thereby disrupting the target from its normal actions.

ACCESSION NUMBER: 2002:113837 BIOSIS  
DOCUMENT NUMBER: PREV200200113837  
TITLE: Method of intracellular binding target molecules.  
AUTHOR(S): Marasco, Wayne A. [Inventor]; **Haseltine, William A.** [Inventor]  
CORPORATE SOURCE: ASSIGNEE: Dana-Farber Cancer Institute, Inc.  
PATENT INFORMATION: US 6329173 December 11, 2001  
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Dec. 11, 2001) Vol. 1253, No. 2.  
<http://www.uspto.gov/web/menu/patdata.html>. e-file.  
CODEN: OGUPE7. ISSN: 0098-1133.

DOCUMENT TYPE: Patent  
LANGUAGE: English  
ENTRY DATE: Entered STN: 30 Jan 2002  
Last Updated on STN: 26 Feb 2002

L2 ANSWER 44 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Beyond chicken soup.  
ACCESSION NUMBER: 2001:563884 BIOSIS  
DOCUMENT NUMBER: PREV200100563884  
TITLE: Beyond chicken soup.  
AUTHOR(S): **Haseltine, William A.**  
SOURCE: Scientific American, (November, 2001) Vol. 285, No. 5, pp. 56-63. print.  
CODEN: SCAMAC. ISSN: 0036-8733.

DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 5 Dec 2001  
Last Updated on STN: 25 Feb 2002

L2 ANSWER 45 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Human DNA ligase III.  
AB A human DNA Ligase III polypeptide and DNA (RNA) encoding such polypeptide and a procedure for producing such polypeptide by recombinant techniques is disclosed. Also disclosed are methods for utilizing such polypeptide via gene therapy for the treatment of disorders associated with a defect in DNA Ligase III. Antagonists against such polypeptides and their use as a therapeutic to destroy unwanted cells are also disclosed. Diagnostic assays to detect mutant DNA Ligase III genes are also disclosed.

ACCESSION NUMBER: 2001:519311 BIOSIS  
DOCUMENT NUMBER: PREV200100519311  
TITLE: Human DNA ligase III.  
AUTHOR(S): Wei, Ying-Fei [Inventor]; Yu, Guo-Liang [Inventor]; **Haseltine, William A.** [Inventor, Reprint author]  
CORPORATE SOURCE: NW. Washington, DC, USA  
ASSIGNEE: Human Genome Sciences, Inc.  
PATENT INFORMATION: US 6284504 September 04, 2001  
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Sep. 4, 2001) Vol. 1250, No. 1. e-file.  
CODEN: OGUPE7. ISSN: 0098-1133.

DOCUMENT TYPE: Patent  
LANGUAGE: English  
ENTRY DATE: Entered STN: 7 Nov 2001

Last Updated on STN: 23 Feb 2002

L2 ANSWER 46 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Method of intracellular binding of target molecules.  
AB The present invention relates to a method by which one can target an undesired target molecule or target antigen, preferably a protein. The method comprises the intracellular expression of an antibody capable of binding to the target. A DNA sequence is delivered to a cell, the DNA sequence contains a sufficient number of nucleotides coding for the portion of an antibody capable of binding to the target operably linked to a promoter that will permit expression of the antibody in the cell(s) of interest. The antibody is then expressed intracellularly and binds to the target, thereby disrupting the target from its normal actions.

ACCESSION NUMBER: 2001:70861 BIOSIS  
DOCUMENT NUMBER: PREV200100070861  
TITLE: Method of intracellular binding of target molecules.  
AUTHOR(S): Marasco, Wayne A. [Inventor]; **Haseltine, William A.** [Inventor]  
CORPORATE SOURCE: ASSIGNEE: Dana-Farber Cancer Institute, Inc.  
PATENT INFORMATION: US 6072036 June 06, 2000  
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (June 6, 2000) Vol. 1235, No. 1. e-file.  
CODEN: OGUPE7. ISSN: 0098-1133.  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
ENTRY DATE: Entered STN: 7 Feb 2001  
Last Updated on STN: 12 Feb 2002

L2 ANSWER 47 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Vector comprising a replication competent HIV-1 provirus and a heterologous gene.  
AB A vector comprising an HIV segment and a heterologous gene segment, which produces a replication competent and an infective HIV virus is disclosed. When the heterologous gene is a marker gene, the spread of the virus can be observed in both in vitro and in vivo systems. The use of this vector in establishing methods for screening anti-viral compounds is also disclosed.

ACCESSION NUMBER: 2000:398269 BIOSIS  
DOCUMENT NUMBER: PREV200000398269  
TITLE: Vector comprising a replication competent HIV-1 provirus and a heterologous gene.  
AUTHOR(S): **Haseltine, William A.** [Inventor, Reprint author]; Terwilliger, Ernest [Inventor]  
CORPORATE SOURCE: Cambridge, MA, USA  
ASSIGNEE: Dana-Farber Cancer Institute  
PATENT INFORMATION: US 6033902 March 07, 2000  
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Mar. 7, 2000) Vol. 1232, No. 1. e-file.  
CODEN: OGUPE7. ISSN: 0098-1133.  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
ENTRY DATE: Entered STN: 20 Sep 2000  
Last Updated on STN: 8 Jan 2002

L2 ANSWER 48 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Vectors containing HIV packaging sequences, packaging defective HIV vectors, and uses thereof.  
AB Packaging defective and packaging proficient HIV vectors are disclosed. These vectors can be used to establish HIV packaging defective cell lines, and to package desired genes. These cell lines can be used in developing a vaccine, HIV antibodies and as part of a system for gene transfer. The packaging proficient vector can be used to target HIV target cells.

ACCESSION NUMBER: 2000:294930 BIOSIS  
DOCUMENT NUMBER: PREV200000294930

TITLE: Vectors containing HIV packaging sequences, packaging defective HIV vectors, and uses thereof.  
AUTHOR(S): Sodroski, Joseph G. [Inventor, Reprint author];  
**Haseltine, William A.** [Inventor]; Poznansky, Mark [Inventor]; Lever, Andrew [Inventor]  
CORPORATE SOURCE: Pinner, UK  
ASSIGNEE: Dana-Farber Cancer Institute, Boston, MA, USA  
PATENT INFORMATION: US 5981276 November 09, 1999  
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Nov. 9, 1999) Vol. 1228, No. 2. e-file.  
CODEN: OGUPE7. ISSN: 0098-1133.  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
ENTRY DATE: Entered STN: 6 Jul 2000  
Last Updated on STN: 7 Jan 2002

L2 ANSWER 49 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Method of intracellular binding of target molecules.  
AB The present invention relates to a method by which one can target an undesired target molecule or target antigen, preferably a protein. The method comprises the intracellular expression of an antibody capable of binding to the target. A DNA sequence is delivered to a cell, the DNA sequence contains a sufficient number of nucleotides coding for the portion of an antibody capable of binding to the target operably linked to a promoter that will permit expression of the antibody in the cell(s) of interest. The antibody is then expressed intracellularly and binds to the target, thereby disrupting the target from its normal actions.

ACCESSION NUMBER: 2000:287930 BIOSIS  
DOCUMENT NUMBER: PREV200000287930  
TITLE: Method of intracellular binding of target molecules.  
AUTHOR(S): Marasco, Wayne A. [Inventor, Reprint author];  
**Haseltine, William A.** [Inventor]  
CORPORATE SOURCE: Cambridge, MA, USA  
ASSIGNEE: Dana-Farber Cancer Institute, Boston, MA, USA  
PATENT INFORMATION: US 5965371 October 12, 1999  
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Oct. 12, 1999) Vol. 1227, No. 2. e-file.  
CODEN: OGUPE7. ISSN: 0098-1133.  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
ENTRY DATE: Entered STN: 6 Jul 2000  
Last Updated on STN: 7 Jan 2002

L2 ANSWER 50 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Discovering genes for new medicines.

ACCESSION NUMBER: 1997:156571 BIOSIS  
DOCUMENT NUMBER: PREV199799455774  
TITLE: Discovering genes for new medicines.  
AUTHOR(S): **Haseltine, William A.**  
CORPORATE SOURCE: Human Genome Sci., Rockville, MD, USA  
SOURCE: Scientific American, (1997) Vol. 276, No. 3, pp. 92-97.  
CODEN: SCAMAC. ISSN: 0036-8733.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 15 Apr 1997  
Last Updated on STN: 15 Apr 1997

L2 ANSWER 51 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI The application of genomics to the creation of new pharmaceutical products.

ACCESSION NUMBER: 1996:148685 BIOSIS  
DOCUMENT NUMBER: PREV199698720820  
TITLE: The application of genomics to the creation of new pharmaceutical products.

AUTHOR(S): **Haseltine, William A.**  
CORPORATE SOURCE: Human Genome Sci., 9410 Key West Ave., Rockville, MD 20850, USA  
SOURCE: AAAS Annual Meeting and Science Innovation Exposition, (1996) Vol. 162, No. 0, pp. A2.  
Meeting Info.: 1996 AAAS Annual Meeting and Science Innovation Exposition: The 162nd National Meeting of the American Association for the Advancement of Science. Baltimore, Maryland, USA. February 8-13, 1996.  
DOCUMENT TYPE: Conference; (Meeting)  
Conference; Abstract; (Meeting Abstract)  
LANGUAGE: English  
ENTRY DATE: Entered STN: 3 Apr 1996  
Last Updated on STN: 26 Apr 1996

L2 ANSWER 52 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Animal model for the therapy of acquired immunodeficiency syndrome with reverse transcriptase inhibitors.  
AB The reverse transcriptase (RT) of the human immunodeficiency virus type 1 (HIV-1) is the major target for antiretroviral therapy of the acquired immunodeficiency syndrome (AIDS). While some inhibitors exhibit activity against most retroviral RTs, others are specific for the HIV-1 enzyme. To develop an animal model for the therapy of the HIV-1 infection with RT inhibitors, the RT of the simian immunodeficiency virus (SIV) was replaced by the RT of HIV-1. Macaques infected with this SIV/HIV-1 hybrid virus developed AIDS-like symptoms and pathology. The HIV-1-specific RT inhibitor LY300046 cnddot HCl, but not zidovudine (3'-azido-3'-deoxythymidine (AZT)) delayed the appearance of plasma antigenemia in macaques infected with a high dose of the chimeric virus. Infection of macaques with the chimeric virus seems to be a valuable model to study the in vivo efficacy of new RT inhibitors, the emergence and reversal of drug resistance, the therapy of infections with drug-resistant viruses, and the efficacy of combination therapy.

ACCESSION NUMBER: 1995:480998 BIOSIS  
DOCUMENT NUMBER: PREV199598495298  
TITLE: Animal model for the therapy of acquired immunodeficiency syndrome with reverse transcriptase inhibitors.  
AUTHOR(S): Ueberla, Klaus [Reprint author]; Stahl-Hennig, Christiane; Boettiger, Disa; Maetz-Rensing, Kerstin; Kaup, Franz J.; Li, John; **Haseltine, William A.**; Fleckenstein, Bernhard; Hunsmann, Gerhard; Oeberg, Bo; Sodroski, Joseph  
CORPORATE SOURCE: Inst. Virol., Univ. Erlangen-Nuernberg, Schlossgarten 4, D-91054 Erlangen, Germany  
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1995) Vol. 92, No. 18, pp. 8210-8214.  
CODEN: PNASA6. ISSN: 0027-8424.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 9 Nov 1995  
Last Updated on STN: 9 Nov 1995

L2 ANSWER 53 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Molecular cloning and expression of human cDNAs encoding a novel DNA ligase IV and DNA ligase III, an enzyme active in DNA repair and recombination.  
AB Three distinct DNA ligases, I to III, have been found previously in mammalian cells, but a cloned cDNA has been identified only for DNA ligase I, an essential enzyme active in DNA replication. A short peptide sequence conserved close to the C terminus of all known eukaryotic DNA ligases was used to search for additional homologous sequences in human cDNA libraries. Two different incomplete cDNA clones that showed partial homology to the conserved peptide were identified. Full-length cDNAs were obtained and expressed by in vitro transcription and translation. The



103-kDa product of one cDNA clone formed a characteristic complex with the XRCC1 DNA repair protein and was identical with the previously described DNA ligase III. DNA ligase III appears closely related to the smaller DNA ligase II. The 96-kDa in vitro translation product of the second cDNA clone was also shown to be an ATP-dependent DNA ligase. A fourth DNA ligase (DNA ligase IV) has been purified from human cells and shown to be identical to the 96-kDa DNA ligase by unique agreement between mass spectrometry data on tryptic peptides from the purified enzyme and the predicted open reading frame of the cloned cDNA. The amino acid sequences of DNA ligases III and IV share a related active-site motif and several short regions of homology with DNA ligase I, other DNA ligases, and RNA capping enzymes. DNA ligases III and IV are encoded by distinct genes located on human chromosomes 17q11.2-12 and 13q33-34, respectively.

ACCESSION NUMBER: 1995:298466 BIOSIS  
 DOCUMENT NUMBER: PREV199598312766  
 TITLE: Molecular cloning and expression of human cDNAs encoding a novel DNA ligase IV and DNA ligase III, an enzyme active in DNA repair and recombination.  
 AUTHOR(S): Wei, Ying-Fei; Robins, Peter; Carter, Kenneth; Caldecott, Keith; Pappin, Darryl J. C.; Yu, Guo-Liang; Wang, Rui-Ping; Shell, Brenda K.; Nash, Rachel A.; Schar, Primo; Barnes, Deborah E.; **Haseltine, William A.**; Lindahl, Tomas [Reprint author]  
 CORPORATE SOURCE: Imperial Cancer Res. Fund, Clare Hall Lab., South Mimms, Hertfordshire EN6 3LD, UK  
 SOURCE: Molecular and Cellular Biology, (1995) Vol. 15, No. 6, pp. 3206-3216.  
 CODEN: MCEBD4. ISSN: 0270-7306.  
 DOCUMENT TYPE: Article  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 11 Jul 1995  
 Last Updated on STN: 2 Aug 1995

L2 ANSWER 54 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 TI Functional analysis of the phosphorylation sites on the human immunodeficiency virus type 1 Vpu protein.  
 AB The human immunodeficiency virus type 1 (HIV-1)-encoded vpu product is a small class 1 integral membrane protein that is phosphorylated by the ubiquitous casein kinase II (CKII) in HIV-1-infected cells. The Vpu protein facilitates the release of budding virions from the surface of infected cells and delays the rate of syncytium formation. In this study, we investigated the role of phosphorylation in the biological activity of Vpu. Our results show that phosphorylation of Vpu occurs on serine residues at positions 52 and 56 located in a highly conserved dodecapeptide sequence. Mutation of either Ser 56, or both Ser 52 and Ser 56 impaired the ability of Vpu to delay the rate of syncytium formation while retaining virion release activity at levels comparable to vpu+ proviruses. Flow cytometry analysis indicates that the relative amounts of envelope glycoprotein gp120 expressed at the surface of cells transfected with these vpu mutant proviruses was two- to threefold greater than that observed on cells transfected with a vpu+ provirus. This increased expression of gp120 at the cell surface may explain the more rapid onset of syncytium formation observed in cell transfected with vpu mutant proviruses. These results suggest that Vpu-facilitated virion release and delayed cytopathic effect are the consequence of two distinct functional activities of the protein.

ACCESSION NUMBER: 1995:182902 BIOSIS  
 DOCUMENT NUMBER: PREV199598197202  
 TITLE: Functional analysis of the phosphorylation sites on the human immunodeficiency virus type 1 Vpu protein.  
 AUTHOR(S): Friborg, Jacques; Ladha, Azim; Gottlinger, Heinrich; **Haseltine, William A.**; Cohen, Eric A. [Reprint author]  
 CORPORATE SOURCE: Dep. Microbiol. Immunol., Fac. Med., Univ. Montreal, CP6128

SOURCE: Station A, Montreal, PQ H3C 3J7, Canada  
Journal of Acquired Immune Deficiency Syndromes and Human  
Retrovirology, (1995) Vol. 8, No. 1, pp. 10-22.  
ISSN: 1077-9450.

DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 26 Apr 1995  
Last Updated on STN: 9 Jun 1995

L2 ANSWER 55 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Characterization of an IL-2 dependent human T cell leukemia virus type I  
(HTLV-I) infected cell line: A system for studying HTLV-I mediated  
transformation.

AB The retrovirus Human T cell Leukemia Virus type I (HTLV-I) is the  
causative agent of Adult T cell Leukemia Lymphoma (ATLL) and is associated  
with HTLV-1 Myelopathy. HTLV-I mediated transformation of CD4+ T cells,  
during the course of ATLL, is poorly understood. It has been suggested  
that HTLV-I is responsible for the immortalization of infected cells, but  
transformation is dependent on secondary events. To investigate this  
hypothesis, we have isolated an HTLV-I infected T cell line that is  
dependent on IL-2 for growth in tissue culture. Further, a subclone of  
this cell line that is able to grow in the absence of IL-2 has been  
isolated. Both cell lines have identical TCR chain rearrangements and  
cell surface markers. Each cell line produces viral mRNAs and proteins.  
Finally, both of these cell lines are sensitive to rapamycin and  
cyclosporin A regardless of the presence of IL-2. We propose that this  
system will provide a unique opportunity to study transformation to IL-2  
independence in HTLV-1 infected cells.

ACCESSION NUMBER: 1995:127668 BIOSIS  
DOCUMENT NUMBER: PREV199598141968  
TITLE: Characterization of an IL-2 dependent human T cell leukemia  
virus type I (HTLV-I) infected cell line: A system for  
studying HTLV-I mediated transformation.

AUTHOR(S): Rohwer, Forest; Macmaster, William; **Haseltine, William**  
**A.**; Tsoukas, Constantine; McGuire, Kathleen L.  
[Reprint author]

CORPORATE SOURCE: Dep. Biol., Mol. Biol. Inst., San Diego State Univ., San  
Diego, CA 92182, USA

SOURCE: International Journal of Oncology, (1994) Vol. 5, No. 5,  
pp. 1163-1169.  
ISSN: 1019-6439.

DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 29 Mar 1995  
Last Updated on STN: 29 Mar 1995

L2 ANSWER 56 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Integrase mutants of human immunodeficiency virus type 1 with a specific  
defect in integration.

AB A previous genetic analysis of the human immunodeficiency virus type 1  
integrase protein failed to identify single amino acid substitutions that  
only block the integration of viral DNA (C.-G. Shin, B. Taddeo, W. A.  
Haseltine, and C. M. Farnet, J. Virol. 68:1633-1642, 1994). Additional  
substitutions of amino acids that are highly conserved among retroviral  
integrases were constructed in human immunodeficiency virus type I and  
analyzed for their effects on viral protein synthesis and processing,  
virion morphology, and viral DNA synthesis and integration in an attempt  
to identify mutants with a specific defect in integration. Four single  
amino acid substitutions resulted in replication defective viruses.  
Conservative, single amino acid substitutions of the two invariant  
aspartic acid residues found in all retroviral integrases prevented the  
integration of viral DNA and had no detectable effect on the other stages  
in the viral replication cycle, indicating that these mutants exhibited a  
specific defect in integration. Mutations at two positions, S-81 and

P-109, blocked the integration of viral DNA but also resulted in the production of viral particles that exhibited reduced reverse transcriptase activity, suggesting additional defects in viral replication. Substitution of the highly conserved amino acid T66 had no effect on viral replication in a CD4+ human T-cell line. This analysis extends the range of possible phenotypes that may be produced by single amino acid substitutions in conserved residues of the integrase protein.

ACCESSION NUMBER: 1995:34040 BIOSIS  
DOCUMENT NUMBER: PREV199598048340  
TITLE: Integrase mutants of human immunodeficiency virus type 1 with a specific defect in integration.  
AUTHOR(S): Taddeo, Brunella; **Haseltine, William A.**; Farnet, Chris M. [Reprint author]  
CORPORATE SOURCE: Div. Human Retrovirol., Dana-Farber Cancer Inst., Boston, MA 02115, USA  
SOURCE: Journal of Virology, (1994) Vol. 68, No. 12, pp. 8401-8405. CODEN: JOVIAM. ISSN: 0022-538X.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 25 Jan 1995  
Last Updated on STN: 26 Jan 1995

L2 ANSWER 57 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Mutations of two PMS homologues in hereditary nonpolyposis colon cancer.  
AB Hereditary nonpolyposis colorectal cancer (HNPCC) is one of man's commonest hereditary diseases'. Several studies have implicated a defect in DNA mismatch repair in the pathogenesis of this disease. In particular, hMSH2 and hMLH1 homologues of the bacterial DNA mismatch repair genes mutS and mutL, respectively, were shown to be mutated in a subset of HNPCC cases. Here we report the nucleotide sequence, chromosome localization and mutational analysis of hPMS1 and hPMS2, two additional homologues of the prokaryotic mutL gene. Both hPMS1 and hPMS2 were found to be mutated in the germline of HNPCC patients. This doubles the number of genes implicated in HNPCC and may help explain the relatively high incidence of this disease.

ACCESSION NUMBER: 1994:482690 BIOSIS  
DOCUMENT NUMBER: PREV199497495690  
TITLE: Mutations of two PMS homologues in hereditary nonpolyposis colon cancer.  
AUTHOR(S): Nicolaides, Nicholas C.; Papadopoulos, Nickolas; Liu, Bo; Wei, Ying-Fel; Carter, Kenneth C.; Ruben, Steven M.; Rosen, Craig A.; **Haseltine, William A.**; Fleischmann, Robert D.  
CORPORATE SOURCE: Inq.; Kenneth W. Kinzler, Johns Hopkins Oncol. Cent., Baltimore, MD 21231, USA  
SOURCE: Nature (London), (1994) Vol. 371, No. 6492, pp. 75-80. CODEN: NATUAS. ISSN: 0028-0836.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 9 Nov 1994  
Last Updated on STN: 9 Nov 1994

L2 ANSWER 58 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Mutation of mutL homolog in hereditary colon cancer.  
AB Some cases of hereditary nonpolyposis colorectal cancer (HNPCC) are due to alterations in a mutS-related mismatch repair gene. A search of a large database of expressed sequence tags derived from random complementary DNA clones revealed three additional human mismatch repair genes, all related to the bacterial mutL gene. One of these genes (hMLH1) resides on chromosome 3p21, within 1 centimorgan of markers previously linked to cancer susceptibility in HNPCC kindreds. Mutations of hMLH1 that would disrupt the gene product were identified in such kindreds, demonstrating that this gene is responsible for the disease. These results suggest that defects in any of several mismatch repair genes can cause HNPCC.

ACCESSION NUMBER: 1994:228198 BIOSIS  
 DOCUMENT NUMBER: PREV199497241198  
 TITLE: Mutation of mutL homolog in hereditary colon cancer.  
 AUTHOR(S): Papadopoulos, Nickolas; Nicoladies, Nicholas C.; Wei, Ying-Fei; Ruben, Steven M.; Carter, Kenneth C.; Rosen, Craig A.; **Haseltine, William A.**; Fleischmann, Robert D.; Fraser, Claire M.; Adams, Mark D.; Venter, J. Craig; Hamilton, Stanley R.; Petersen, Gloria M.  
 CORPORATE SOURCE: Johns Hopkins Oncol. Cent., Baltimore, MD 21231, USA  
 SOURCE: Science (Washington D C), (1994) Vol. 263, No. 5153, pp. 1625-1629.  
 CODEN: SCIEAS. ISSN: 0036-8075.  
 DOCUMENT TYPE: Article  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 24 May 1994  
 Last Updated on STN: 24 May 1994

L2 ANSWER 59 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 TI Role of the matrix protein in the virion association of the human immunodeficiency virus type 1 envelope glycoprotein.  
 AB The matrix (MA) protein of human immunodeficiency virus type 1 (HIV-1) forms an inner coat directly underneath the lipid envelope of the virion. The outer surface of the lipid envelope surrounding the capsid is coated by the viral Env glycoproteins. We report here that the HIV-1 capsid-Env glycoprotein association is very sensitive to minor alterations in the MA protein. The results indicate that most of the MA domain of the Gag precursor, except for its carboxy terminus, is essential for this association. Viral particles produced by proviruses with small missense or deletion mutations in the region coding for the amino-terminal 100 amino acids of the MA protein lacked both the surface glycoprotein gp120 and the transmembrane glycoprotein gp41, indicating a defect at the level of Env glycoprotein incorporation. Alterations at the carboxy terminus of the MA domain had no significant effect on the levels of particle-associated Env glycoprotein or on virus replication. The presence of HIV-1 MA protein sequences was sufficient for the stable association of HIV-1 Env glycoprotein with hybrid particles that contain the capsid (CA) and nucleocapsid (NC) proteins of visna virus. The association of HIV-1 Env glycoprotein with the hybrid particles was dependent upon the presence of the HIV-1 MA protein domain, as HIV-1 Env glycoprotein was not efficiently recruited into virus particles when coexpressed with authentic visna virus Gag proteins.

ACCESSION NUMBER: 1994:169996 BIOSIS  
 DOCUMENT NUMBER: PREV199497182996  
 TITLE: Role of the matrix protein in the virion association of the human immunodeficiency virus type 1 envelope glycoprotein.  
 AUTHOR(S): Dorfman, Tatyana; Mammano, Fabrizio; **Haseltine, William A.**; Goettlinger, Heinrich G. [Reprint author]  
 CORPORATE SOURCE: Div. Human Retrovirology, Dana-Farber Cancer Inst., Jimmy Fund Build., Room 824, 44 Binney St., Boston, MA 02115, USA  
 SOURCE: Journal of Virology, (1994) Vol. 68, No. 3, pp. 1689-1696.  
 CODEN: JOVIAM. ISSN: 0022-538X.  
 DOCUMENT TYPE: Article  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 8 Apr 1994  
 Last Updated on STN: 8 Apr 1994

L2 ANSWER 60 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 TI Requirement of the Pr55-gag precursor for incorporation of the Vpr product into human immunodeficiency virus type 1 viral particles.  
 AB The human immunodeficiency virus type 1 (HIV-1) particles consists of two molecules of genomic RNA as well as molecules originating from gag, pol, and env products, all synthesized as precursor proteins. The 96-amino-acid Vpr protein, the only virion-associated HIV-1 regulatory protein, is not part of the virus polyprotein precursors, and its

incorporation into virus particles must occur by way of an interaction with a component normally found in virions. To investigate the mechanism of incorporation of Vpr into the HIV-1 virion, Vpr- proviral DNA constructs harboring mutations or deletions in specific virion-associated gene products were cotransfected with Vpr expressor plasmids in COS cells. Virus released from the transfected cells was tested for the presence of Vpr by immunoprecipitation with Vpr-specific antibodies. The results of these experiments show that Vpr is trans-incorporated into virions but at a lower efficiency than when Vpr is expressed from a proviral construct. The minimal viral genetic information necessary for Vpr incorporation was a deleted provirus encoding only the pr55-gag polyprotein precursor. Incorporation of Vpr requires the expression but not the processing of gag products and is independent of pol and env expression. Direct interaction of Vpr with the Pr55-gag precursor protein was demonstrated by coprecipitation experiments with gag product-specific antibodies. Overall, these results indicate that HIV-1 Vpr is incorporated into the nascent virion through an interaction with the Gag precursor polyprotein and demonstrate a novel mechanism by which viral protein can be incorporated into virus particles.

ACCESSION NUMBER: 1994:169990 BIOSIS  
DOCUMENT NUMBER: PREV199497182990  
TITLE: Requirement of the Pr55-gag precursor for incorporation of the Vpr product into human immunodeficiency virus type 1 viral particles.  
AUTHOR(S): Lavallee, Claude; Yao, Xiao Jian; Ladha, Azim; Goettlinger, Heinrich; **Haseltine, William A.**; Cohen, Eric A.  
[Reprint author]  
CORPORATE SOURCE: Lab. de Retrovirologie Humaine, Dep. de Microbiologie et Immunologie, Fac. de Med., Univ. de Montreal, Montreal, PQ H3C 3J7, Canada  
SOURCE: Journal of Virology, (1994) Vol. 68, No. 3, pp. 1926-1934.  
CODEN: JOVIAM. ISSN: 0022-538X.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 8 Apr 1994  
Last Updated on STN: 8 Apr 1994

L2 ANSWER 61 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Genetic analysis of the human immunodeficiency virus type 1 integrase protein.  
AB Single-amino-acid changes in a highly conserved central region of the human immunodeficiency virus type 1 (HTV-1) integrase protein were analyzed for their effects on viral protein synthesis, virion morphogenesis, and viral replication. Alteration of two amino acids that are invariant among retroviral integrases, D116 and E152 of HIV-1, as well as a mutation of the highly conserved amino acid S147 blocked viral replication in two CD4+ human T-cell lines. Mutations of four other highly conserved amino acids in the region had no detectable effect on viral replication, whereas mutations at two positions, N117 and Y143, resulted in viruses with a delayed-replication phenotype. Defects in virion precursor polypeptide processing, virion morphology, or viral DNA synthesis were observed for all of the replication-defective mutants, indicating that changes in integrase can have pleiotropic effects on viral replication.

ACCESSION NUMBER: 1994:169966 BIOSIS  
DOCUMENT NUMBER: PREV199497182966  
TITLE: Genetic analysis of the human immunodeficiency virus type 1 integrase protein.  
AUTHOR(S): Shin, Cha-Gyun; Taddeo, Brunella; **Haseltine, William A.**; Farnet, Chris M. [Reprint author]  
CORPORATE SOURCE: Dana-Farber Cancer Inst., 44 Binney St., JFB824, Boston, MA 02115, USA  
SOURCE: Journal of Virology, (1994) Vol. 68, No. 3, pp. 1633-1642.  
CODEN: JOVIAM. ISSN: 0022-538X.

DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 8 Apr 1994  
Last Updated on STN: 11 May 1994

L2 ANSWER 62 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Mapping of functionally important residues of a cysteine-histidine box in  
the human immunodeficiency virus type 1 nucleocapsid protein.  
AB The human immunodeficiency virus type 1 (HIV-1) nucleocapsid protein  
contains two copies of a sequence motif, the cysteine-histidine box, that  
is conserved among retroviruses. To identify the functionally relevant  
positions of a cysteine-histidine box, each amino acid in the proximal  
copy of the motif was individually substituted by site-directed  
mutagenesis. Mutations at 5 of 14 positions abolished virus replication  
and reduced the viral RNA content of mutant particles to between 10 and  
20% of parental levels. Mutations at other positions had either no or  
only a minor effect on virus replication and virion RNA content. In vitro  
binding of RNA to bacterially expressed mutant Pr55-gag polyprotein  
correlated well with the effects of the mutations on particle-associated  
viral RNA levels. The two different copies of the motif in the HIV-1  
nucleocapsid protein are not functionally equivalent, since the conversion  
of the proximal motif to an exact copy of the distal motif results in a  
defect in virus replication and a reduction in the viral RNA content of  
mutant particles. The simultaneous substitution of functionally relevant  
positions in both motifs led to a significant decline in gag protein  
export, indicating that the nucleocapsid domain of the gag precursor is  
also required for efficient assembly or release of the virion.

ACCESSION NUMBER: 1993:507511 BIOSIS  
DOCUMENT NUMBER: PREV199396131518  
TITLE: Mapping of functionally important residues of a  
cysteine-histidine box in the human immunodeficiency virus  
type 1 nucleocapsid protein.  
AUTHOR(S): Dorfman, Tatyana; Luban, Jeremy; Goff, Stephen P.;  
**Haseltine, William A.**; Goettlinger, Heinrich G.  
[Reprint author]  
CORPORATE SOURCE: Div. Human Retrovirology, Dana-Farber Cancer Inst., Harvard  
Med. Sch., Boston, MA 02115, USA  
SOURCE: Journal of Virology, (1993) Vol. 67, No. 10, pp. 6159-6169.  
CODEN: JOVIAM. ISSN: 0022-538X.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 5 Nov 1993  
Last Updated on STN: 5 Nov 1993

L2 ANSWER 63 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Design, intracellular expression, and activity of a human anti-human  
immunodeficiency virus type 1 gp120 single-chain antibody.  
AB A single-chain antibody, derived from a human monoclonal antibody that  
recognizes the CD4 binding region of the human immunodeficiency virus type  
1 (HIV-1) envelope protein, has been designed for intracellular expression  
in eukaryotic cells. The single-chain antibody is composed of an  
immunoglobulin heavy-chain leader sequence and heavy and light-chain  
variable regions that are joined by an interchain linker. The antibody is  
stably expressed and retained in the endoplasmic reticulum and is not  
toxic to the cells. The antibody binds to the envelope protein within the  
cell and inhibits processing of the envelope precursor and syncytia  
formation. The infectivity of the HIV-1 particles produced by cells that  
express the single-chain antibody is substantially reduced. These studies  
illustrate the feasibility of designing antibodies that bind and  
inactivate molecules intracellularly. Antibodies that act on target  
molecules within cells should provide a useful tool for research as well  
as for control of infectious and other diseases.

ACCESSION NUMBER: 1993:454762 BIOSIS  
DOCUMENT NUMBER: PREV199396099662

TITLE: Design, intracellular expression, and activity of a human anti-human immunodeficiency virus type 1 gp120 single-chain antibody.  
AUTHOR(S): Marasco, Wayne A. [Reprint author]; **Haseltine, William A.**; Chen, Siyi  
CORPORATE SOURCE: Dep. Med., Dana-Farber Cancer Inst., Harvard Med. Sch., Harvard Sch. Public Health, 44 Binney St., Boston, MA 02115, USA  
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1993) Vol. 90, No. 16, pp. 7889-7893.  
CODEN: PNASA6. ISSN: 0027-8424.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 5 Oct 1993  
Last Updated on STN: 5 Oct 1993

L2 ANSWER 64 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Vpu protein of human immunodeficiency virus type 1 enhances the release of capsids produced by gag gene constructs of widely divergent retroviruses.  
AB The Vpu protein of human immunodeficiency virus type 1 facilitates the release of virus particles from the surface of infected cells. The ability of the Vpu protein to facilitate release of Gag proteins from retroviruses that lack a Vpu-like protein was examined. The results of these experiments show that Vpu significantly increases the release of the Gag proteins of human immunodeficiency virus type 2, visna virus, and Moloney murine leukemia virus from HeLa cells. The results indicate that Vpu-mediated enhancement of particle release requires neither amino-terminal myristoylation of the Gag precursor nor cleavage of the Gag precursor by the viral protease. The results raise the possibility that Vpu modifies a cellular pathway common to the release of all retroviruses from the cell surface.

ACCESSION NUMBER: 1993:432278 BIOSIS  
DOCUMENT NUMBER: PREV199396086903  
TITLE: Vpu protein of human immunodeficiency virus type 1 enhances the release of capsids produced by gag gene constructs of widely divergent retroviruses.  
AUTHOR(S): Gottlinger, Heinrich G. [Reprint author]; Dorfman, Tatyana [Reprint author]; Cohen, Eric A.; **Haseltine, William A.** [Reprint author]  
CORPORATE SOURCE: Div. Human Retrovirol., Dana-Farber Cancer Inst., 44 Binney St., Boston, MA 02115, USA  
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1993) Vol. 90, No. 15, pp. 7381-7385.  
CODEN: PNASA6. ISSN: 0027-8424.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 22 Sep 1993  
Last Updated on STN: 22 Sep 1993

L2 ANSWER 65 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Early molecular replication of human immunodeficiency virus type 1 in cultured-blood-derived T helper dendritic cells.  
AB The rate and efficiency of key steps in the life cycle of the human immunodeficiency virus type 1 was examined in three primary cell types, T cells, monocytes, and T helper dendritic cells using the same quantity of virus involved and same cell number. The results show that viral DNA synthesis proceeds much more rapidly and efficiently in primary T helper dendritic cell populations than in primary T cell and monocyte populations. The increased rate of virus DNA synthesis is attributable either to an increase in the efficiency and the rate of uptake of the virus particles by the T helper dendritic cells, as compared with that in other cell types, or to an increased efficiency and rate of viral DNA

synthesis in the T helper dendritic cells. In the subsequent phase of viral expression the appearance of spliced viral mRNA products also occur more rapidly in cultures of primary-blood-derived T helper dendritic cells than is the case in primary T cells and monocytes. The increased efficiency of the early steps of HIV-1 replication in primary-blood-derived T helper dendritic cells than in other blood-derived mononuclear cells raises the possibility that these cells play a central role in HIV-1 infection and pathogens.

ACCESSION NUMBER: 1993:367258 BIOSIS  
DOCUMENT NUMBER: PREV199396052933  
TITLE: Early molecular replication of human immunodeficiency virus type 1 in cultured-blood-derived T helper dendritic cells.  
AUTHOR(S): Langhoff, Erik [Reprint author]; Kalland, Karl H.;  
**Haseltine, William A.**  
CORPORATE SOURCE: Div. Human Retrovirol., Dana-Farber Cancer Inst., 44 Binney St., Boston, MA 02115, USA  
SOURCE: Journal of Clinical Investigation, (1993) Vol. 91, No. 6, pp. 2721-2726.  
CODEN: JCINAO. ISSN: 0021-9738.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 6 Aug 1993  
Last Updated on STN: 6 Aug 1993

L2 ANSWER 66 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI A possible role of dendritic cells in HIV-1 replication and transmission.  
ACCESSION NUMBER: 1993:353170 BIOSIS  
DOCUMENT NUMBER: PREV199345036595  
TITLE: A possible role of dendritic cells in HIV-1 replication and transmission.  
AUTHOR(S): Langhoff, Erik; **Haseltine, William A.**  
CORPORATE SOURCE: Div. Hum. Retrovirol., Dana-Farber Cancer Inst., Boston, MA, USA  
SOURCE: Koff, W. C. [Editor]; Wong-Staal, F. [Editor]; Kennedy, R. C. [Editor]. AIDS Res. Rev., (1993) pp. 59-71. AIDS Research Reviews.  
Publisher: Marcel Dekker, Inc., 270 Madison Avenue, New York, New York 10016, USA; Marcel Dekker, Inc., Basel, Switzerland. Series: AIDS Research Reviews.  
CODEN: ARRVEZ. ISSN: 1056-1080. ISBN: 0-8247-9045-6.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 31 Jul 1993  
Last Updated on STN: 31 Jul 1993

L2 ANSWER 67 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Effect of nef alleles on replication of human immunodeficiency virus type 1.  
AB The effect of multiple alleles of nef of the human immunodeficiency virus type 1 (HIV-1) on virus replication was examined. Nef alleles used include some derived from isolates of virus passaged in tissue culture as well as other obtained by direct cloning of viral DNA from tissues of infected patients. The effect of nef on virus replication was evaluated in the context of a derivative of the HXB2 provirus shown previously to require nef for rapid growth in CD4+ human T cell lines and in primary peripheral blood mononuclear cells. The results of the experiments show that in this genetic context all of the studied viruses carrying nef alleles that express stable Nef proteins replicate more rapidly than do their, otherwise isogenic, nef-defective counterparts. Two of the nef alleles derived from primary tissues produce unstable proteins. These studies demonstrate that naturally occurring nef alleles can increase the rate of virus replication in both primary peripherals blood mononuclear cells and in a CD4+ T cell line. The results also demonstrate that functional variation exists among naturally occurring nef alleles.



ACCESSION NUMBER: 1993:273958 BIOSIS  
 DOCUMENT NUMBER: PREV199396004183  
 TITLE: Effect of nef alleles on replication of human immunodeficiency virus type 1.  
 AUTHOR(S): Zazopoulos, Emmanuel; **Haseltine, William A.**  
 [Reprint author]  
 CORPORATE SOURCE: Div. Human Retrovirol., Dana-Farber Cancer Inst., 44 Binney St., Boston, MA 02115, USA  
 SOURCE: Virology, (1993) Vol. 194, No. 1, pp. 20-27.  
 CODEN: VIRLAX. ISSN: 0042-6822.  
 DOCUMENT TYPE: Article  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 9 Jun 1993  
 Last Updated on STN: 9 Jun 1993

L2 ANSWER 68 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 TI RNA tumor viruses.

ACCESSION NUMBER: 1993:238916 BIOSIS  
 DOCUMENT NUMBER: PREV199344112116  
 TITLE: RNA tumor viruses.  
 AUTHOR(S): Fine, Howard A. [Reprint author]; **Haseltine, William A.**  
 CORPORATE SOURCE: Div. Clin. Oncol., Dana-Farber Cancer Inst., Boston, MA, USA  
 SOURCE: Holland, J. F. [Editor]; Freii, E., III [Editor]; Bast, R. C., Jr. [Editor]; Kufe, D. W. [Editor]; Morton, D. L. [Editor]; Weichselbaum, R. R. [Editor]. (1993) pp. 2) 265-282. Cancer medicine, Third edition, Vols. 1 and 2. Publisher: Lea and Febiger, 200 Chesterfield Parkway, Malvern, Pennsylvania 19355, USA; Lea and Febiger, London, England, UK.  
 ISBN: 0-8121-1422-1.  
 DOCUMENT TYPE: Article  
 General Review; (Literature Review)  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 15 May 1993  
 Last Updated on STN: 15 May 1993

L2 ANSWER 69 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 TI The NF-kappa-B p65 promoter.

AB The promoter of the human gene encoding the p65 subunit of the transcription factor NIF-kB was cloned and the nucleotide sequence determined. The p65 promoter lacks both TATA and CCAAT consensus sequences. The p65 promoter contains three consensus binding sites of the transcription factor SP1. In contrast to the promoter of the p50 subunit of NF-KB, no sequences predicted to bind NF-KB are present in the p65 promoter. Phorbol ester (PMA) and phytohemagglutinin (PHA) treatment of Jurkat cells did not activated the p65 promoter in transient transfection experiments. Using different deletion mutants of the p65 promoter, essential promoter elements were mapped.

ACCESSION NUMBER: 1993:228529 BIOSIS  
 DOCUMENT NUMBER: PREV199395119704  
 TITLE: The NF-kappa-B p65 promoter.  
 AUTHOR(S): Ueberla, Klaus; Lu, Yichen; Chung, Eugene; **Haseltine, William A.** [Reprint author]  
 CORPORATE SOURCE: Dana-Farber Cancer Inst., JF824, 44 Binney St., Boston, MA 02115, USA  
 SOURCE: Journal of Acquired Immune Deficiency Syndromes, (1993) Vol. 6, No. 3, pp. 227-230.  
 CODEN: JAISSET. ISSN: 0894-9255.  
 DOCUMENT TYPE: Article  
 LANGUAGE: English  
 OTHER SOURCE: Genbank-LO1459  
 ENTRY DATE: Entered STN: 7 May 1993

Last Updated on STN: 7 May 1993

L2 ANSWER 70 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI The effect of vpu on HIV-1-induced syncytia formation.  
AB To investigate the role of vpu in the cytopathicity of human immunodeficiency type 1 (HIV-1), the MT4 CD4+ T-cell line was infected with viruses that were isogenic except for their ability to produce the vpu protein. The experiments described here demonstrate that expression of vpu reduces HIV-1 cytopathic effects by decreasing the rate of syncytia formation. By reducing the concentration of gp120 at the cell surface, vpu limits cell killing by syncytia formation.

ACCESSION NUMBER: 1993:214203 BIOSIS  
DOCUMENT NUMBER: PREV199395115428  
TITLE: The effect of vpu on HIV-1-induced syncytia formation.  
AUTHOR(S): Yao, Xiao Jian; Garzon, Simon; Boisvert, Francoise; **Haseltine, William A.**; Cohen, Eric A. [Reprint author]  
CORPORATE SOURCE: Lab. de Retrovirol. Hum., Dep. Microbiol. Immunol., Fac. Med., Univ. Montreal, Montreal, PQ H3C 3J7, Canada  
SOURCE: Journal of Acquired Immune Deficiency Syndromes, (1993) Vol. 6, No. 2, pp. 135-141.  
CODEN: JAISSET. ISSN: 0894-9255.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 23 Apr 1993  
Last Updated on STN: 23 Apr 1993

L2 ANSWER 71 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Disulfide bond formation in the human immunodeficiency virus type 1 Nef protein.  
AB Substitution of alanine for cysteine residues of the human immunodeficiency virus type 1 LAI (BRU) and ELI Nef proteins was used to determine pairing of the cysteine residues present in each protein. The results show that under nonreducing conditions, alternative pairing of the cysteines occurs. The preferred pairing of cysteine residues of the LAI and ELI proteins differs. In the experimental system used, viruses carrying the ELI nef allele are found to express Nef proteins which accelerate virus replication. Mutation in critical cysteine residues of the protein reduce the rate of virus replication. In the same system, viruses harboring the LAI nef allele fail to replicate. These observations raise the possibility that differences in the observed biological activity of nef alleles may be attributed, at least in part, to differences in the secondary structure of the proteins.

ACCESSION NUMBER: 1993:214199 BIOSIS  
DOCUMENT NUMBER: PREV199395115424  
TITLE: Disulfide bond formation in the human immunodeficiency virus type 1 Nef protein.  
AUTHOR(S): Zazopoulos, Emmanuel; **Haseltine, William A.** [Reprint author]  
CORPORATE SOURCE: Div. Human Retrovirology, Dana-Farber Cancer Inst., 44 Binney St., Boston, MA 02115, USA  
SOURCE: Journal of Virology, (1993) Vol. 67, No. 3, pp. 1676-1680.  
CODEN: JOVIAM. ISSN: 0022-538X.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 23 Apr 1993  
Last Updated on STN: 24 Apr 1993

L2 ANSWER 72 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Influence of human T-cell leukemia virus type I tax and rex on interleukin-2 gene expression.  
AB The X region of human T-cell leukemia virus type I (HTLV-I) encodes two proteins that regulate viral gene expression. The tax protein is the product of the transactivator gene and has been shown to up-regulate the

expression of some cellular genes controlling T-cell replication, including that of the interleukin-2 (IL-2) T-cell growth hormone and the alpha chain of its receptor (IL-2R). Several studies have shown that tax transactivation of the IL-2R alpha-chain promoter is mediated by binding sites for the transcriptional activator NF-kappa-B, and this mechanism has also been implicated in the tax activation of IL-2 promoter activity. The rex gene product of HTLV-1 regulates viral protein production by influencing mRNA expression and has been implicated in the stabilization of IL-2R alpha-chain mRNA. In the present studies, the ability of the tax and rex proteins to transactivate IL-2 gene expression has been reinvestigated. The ability of the tax protein to transactivate IL-2 promoter activity appears, at least in part, to be mediated by the recognition sequence for a DNA-binding complex known as CD28RC. Consistent with this hypothesis is the observation that tax-mediated activation of IL-2 gene expression is resistant to the immunosuppressive effects of cyclosporin A, a property postulated for the CD28RC binding complex. Unexpectedly, this tax-mediated up-regulation of IL-2 expression is synergized by the presence of the rex protein. These findings demonstrate that transactivation of IL-2 gene expression by tax is augmented by mechanisms distinct from NF-kappa-B and raise the possibility that rex, as well as tax, contributes to the oncogenic capability of HTLV-I by altering the expression of the IL-2 gene in T cells infected with this retrovirus.

ACCESSION NUMBER: 1993:209106 BIOSIS  
DOCUMENT NUMBER: PREV199395110331  
TITLE: Influence of human T-cell leukemia virus type I tax and rex on interleukin-2 gene expression.  
AUTHOR(S): McGuire, Kathleen L. [Reprint author]; Curtiss, Virginia E.; Larson, Erica L.; **Haseltine, William A.**  
CORPORATE SOURCE: Dep. Biol., Coll. Sci., San Diego State Univ., San Diego, CA 92182-0057, USA  
SOURCE: Journal of Virology, (1993) Vol. 67, No. 3, pp. 1590-1599. CODEN: JOVIAM. ISSN: 0022-538X.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 23 Apr 1993  
Last Updated on STN: 23 Apr 1993

L2 ANSWER 73 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Complex-type N-linked oligosaccharides of gp120 from human immunodeficiency virus type 1 contain sulfated N-acetylglucosamine.  
AB The major envelope glycoproteins gp120 and gp41 of human immunodeficiency virus type 1, the causative agent for human AIDS, contain numerous N-linked oligosaccharides. We report here our discovery that N-acetylglucosamine residues within the complex-type N-linked oligosaccharides of both gp120 and its precursor, gp160, are sulfated. When human Molt-3 cells persistently infected with human T-cell leukemia virus III-B were metabolically radiolabeled with 35SO-4, gp160, gp120, and to some extent gp41 were radiolabeled. The 35SO-4-labeled oligosaccharides were quantitatively released by N-glycanase treatment and were bound by immobilized Ricinus communis agglutinin I, a lectin that binds to terminal P-galactosyl residues. The kinetics of release of sulfate upon acid hydrolysis from 35SO-4-labeled gp120 indicate that sulfation occurs in a primary sulfate ester linkage. Methylation analysis of total glycopeptides from Molt-3 cells metabolically radiolabeled with (3H)glucosamine demonstrates that sulfation occurs at the C-6 position of N-acetylglucosamine. Fragmentation of the gp120-derived 35SO-4-labeled glycopeptides by treatment with hydrazine and nitrous acid and subsequent reduction generated galactosyl-anhydromannitol-6-35SO-4, which is the expected reaction product from GlcNAc-6-sulfate within a sulfated lactosamine moiety. Charge analysis of the (3H)galactose- and (3H)glucosamine-labeled glycopeptides from gp120 and gp160 indicates that approximately 14% of the complex-type N-linked oligosaccharides are sulfated.

ACCESSION NUMBER: 1993:148372 BIOSIS  
DOCUMENT NUMBER: PREV199395081172  
TITLE: Complex-type N-linked oligosaccharides of gp120 from human immunodeficiency virus type 1 contain sulfated N-acetylglucosamine.  
AUTHOR(S): Shilatifard, Ali; Merkle, Roberta K.; Helland, Dag E.; Welles, Jacqueline L.; **Haseltine, William A.**; Cummings, Richard D. [Reprint author]  
CORPORATE SOURCE: Dep. Biochem. and Molecular Biol., Univ. Okla. Health Sci. Cent., P.O. Box 26901, 941 S. L. Young Boulevard, Oklahoma City, OK 3104, USA  
SOURCE: Journal of Virology, (1993) Vol. 67, No. 2, pp. 943-952. CODEN: JOVIAM. ISSN: 0022-538X.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 16 Mar 1993  
Last Updated on STN: 17 Mar 1993

L2 ANSWER 74 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Infection of human natural killer (NK) cells with replication-defective human T cell leukemia virus type I provirus: Increased proliferative capacity and prolonged survival of functionally competent NK cells.  
AB Human T-cell leukemia virus type I (HTLV-I) can infect a variety of human cell types, but only T lymphocytes are efficiently immortalized after HTLV-I infection. This study reports an attempt to infect and to immortalize NK cells with HTLV-I. Co-cultivation of freshly isolated NK cells with a HTLV-I-producing T cell line did not result in NK cell infection. However, NK cells activated with an anti-CD16 mAb and co-cultivated with a HTLV-I-producing T cell line were reproducibly infected by HTLV-I. HTLV-I infection was documented in NK cell lines and clones by the detection of defective integrated provirus by both Southern blot and polymerase chain reaction analysis. Although HTLV-I-infected NK cells produced viral proteins, they did not produce infectious viral particles. HTLV-I-infected NK cells have phenotypically indistinguishable from their uninfected counterparts (CD16+, CD2+, CD56+, CD3-). They also retained the ability to mediate both natural and antibody-dependent cell cytotoxicity. The IL-2-dependent proliferation of HTLV-I-infected NK cells was significantly greater than that of uninfected NK cells. The doubling time of this infected population was reduced from 9 days to 3 days, and the overall survival of the culture in the absence of restimulation was extended from 5 wk to 18 wk. Unlike T lymphocytes, HTLV-I-infected NK cells were not immortal, implying a fundamental difference between these two lymphocyte populations.

ACCESSION NUMBER: 1993:118274 BIOSIS  
DOCUMENT NUMBER: PREV199395062374  
TITLE: Infection of human natural killer (NK) cells with replication-defective human T cell leukemia virus type I provirus: Increased proliferative capacity and prolonged survival of functionally competent NK cells.  
AUTHOR(S): Lo, K. M. Steve; Vivier, Eric; Rochet, Nathalie; Dehni, Ghassan; Levine, Herbert; **Haseltine, William A.**; Anderson, Paul [Reprint author]  
CORPORATE SOURCE: Div. Tumor Immunology, Dana-Farber Cancer Inst., 44 Binney St., Boston, Mass. 02115, USA  
SOURCE: Journal of Immunology, (1992) Vol. 149, No. 12, pp. 4101-4108. CODEN: JOIMA3. ISSN: 0022-1767.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 27 Feb 1993  
Last Updated on STN: 27 Feb 1993

L2 ANSWER 75 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Infection of accessory dendritic cells by human immunodeficiency virus

type 1.

ACCESSION NUMBER: 1993:86648 BIOSIS  
DOCUMENT NUMBER: PREV199344040898  
TITLE: Infection of accessory dendritic cells by human immunodeficiency virus type 1.  
AUTHOR(S): Langhoff, Erik; **Haseltine, William A.** [Reprint author]  
CORPORATE SOURCE: Div. Human Retrovirol., Dana-Farber Cancer Inst., Harv. Med. Sch., 44 Binney St., Boston, Mass. 02115, USA  
SOURCE: Journal of Investigative Dermatology, (1992) Vol. 99, No. 5, pp. 89S-94S.  
Meeting Info.: Third International Workshop on Langerhans Cells. Dallas, Texas, USA. December 5-6, 1991.  
CODEN: JIDEAE. ISSN: 0022-202X.  
DOCUMENT TYPE: Article  
Conference; (Meeting)  
LANGUAGE: English  
ENTRY DATE: Entered STN: 1 Feb 1993  
Last Updated on STN: 1 Feb 1993

L2 ANSWER 76 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Ganglioside-induced CD4 endocytosis occurs independent of serine phosphorylation and is accompanied by dissociation of P56-lck.  
AB Gangliosides induce a selective and complete modulation of CD4 from the surface of T cells. CD4 down-modulation occurs by CD4 endocytosis. This process is independent of serine phosphorylation of the cytoplasmic tail of CD4 and does not require the association between the tyrosine protein kinase p56-lck and the cytoplasmic tail of CD4. Ganglioside induced CD4 endocytosis is accompanied by the loss of p56-lck activity associated with CD4. Sequential immunoprecipitation analysis using an anti-CD4 antibody and an anti-p56-lck antiserum showed that this is caused by the dissociation of the enzyme from the cytoplasmic tail of CD4. The kinetics of p56-lck dissociation after ganglioside treatment is identical to that of CD4 endocytosis, suggesting that p56-lck is displaced in the process of endosome formation. The results indicate that CD4 endocytosis alone can cause the dissociation of the p56-lck complex without the requirement for Cd4 phosphorylation.

ACCESSION NUMBER: 1993:74771 BIOSIS  
DOCUMENT NUMBER: PREV199395039271  
TITLE: Ganglioside-induced CD4 endocytosis occurs independent of serine phosphorylation and is accompanied by dissociation of P56-lck.  
AUTHOR(S): Repke, Heinrich [Reprint author]; Barber, Elizabeth; Ulbricht, Stefanie; Buchner, Klaus; Hucho, Ferdinand; Kopp, Richard; Scholz, Hans; Rudd, Christopher E.;  
**Haseltine, William A.**  
CORPORATE SOURCE: Div. Human Retrovirol., Dana-Farber-Cancer Inst., 44 Binney St., Boston, MA 02115, USA  
SOURCE: Journal of Immunology, (1992) Vol. 149, No. 8, pp. 2585-2591.  
CODEN: JOIMA3. ISSN: 0022-1767.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 26 Jan 1993  
Last Updated on STN: 27 Jan 1993

L2 ANSWER 77 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Role of vif in replication of human immunodeficiency virus type 1 in CD4-positive T lymphocytes.  
AB The viral infectivity factor gene vif of human immunodeficiency virus type 1 has been shown to affect the infectivity but not the production of virus particles. In this study, the effect of vif in the context of the HXB2 virus on virus replication in several CD4+ T-cell lines was investigated. vif was found to be required for replication in the CD4+ T-cell lines CEM

and H9 as well as in peripheral blood T lymphocytes. vif was not required for replication in the SupT1, C8166, and Jurkat T-cell lines. The infectivity of vif-defective viruses depended on the cell type in which the virus was produced. In CEM cells, vif was required for production of virus capable of initiating infection in all cell lines studied. vif-defective virus produced by SupT1, C8166, and Jurkat cells and the monkey cell line COS-1 could initiate infection in multiple cell lines, including CEM and H9. These results suggest that vif can compensate for cellular factors required for production of infectious virus particles that are present in some cell lines such as SupT1, C8166, and Jurkat but are absent in others such as CEM and H9 as well as peripheral blood T lymphocytes. The effect of vif was not altered by deletion of the carboxyl terminus of gp41, a proposed target for vif (B. Guy, M. Geist, K. Dott, D. Spehner, M.-P. Kieny, and J.-P. Lecocq, J. Virol. 65:1325-1331, 1991). These studies demonstrate that vif enhances viral infectivity during virus production and also suggest that vif is likely to be important for natural infections.

ACCESSION NUMBER: 1993:34860 BIOSIS  
DOCUMENT NUMBER: PREV199395023060  
TITLE: Role of vif in replication of human immunodeficiency virus type 1 in CD4-positive T lymphocytes.  
AUTHOR(S): Gabuzda, Dana H.; Lawrence, Katharine; Langhoff, Erik; Terwilliger, Ernest; Dorfman, Tatyana; **Haseltine, William A.**; Sodroski, Joseph [Reprint author]  
CORPORATE SOURCE: Division Human Retrovirology, Dana-Farber Cancer Institute, 44 Binney Street, Boston, Mass. 02115, USA  
SOURCE: Journal of Virology, (1992) Vol. 66, No. 11, pp. 6489-6495. CODEN: JOVIAM. ISSN: 0022-538X.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 23 Dec 1992  
Last Updated on STN: 23 Dec 1992

L2 ANSWER 78 OF 78 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
TI Characterization of the cDNA of a broadly reactive neutralizing human anti-gp120 monoclonal antibody.  
AB The F105 mAb, identified in an HIV-1-infected individual, binds to a discontinuous epitope on HIV-1 gp120 envelope glycoprotein, blocks the binding of gp120 to CD4 viral receptor, and neutralizes a broad range of HIV-1 isolates. This study reports the primary nucleotide and deduced amino acid sequences of the rearranged heavy and light chains of the mAb F105. This IgG-1k mAb uses a V-H gene member of the V-H4 gene family (V71-4) and is productively rearranged with a D-D fusion product of the dlr4 and da4 germline D-H genes and the J-H5 gene. This rearrangement heavy chain gene expresses the V-H4-HV2a idiotope, which is seen in human monoclonal IgM colde agglutinins. The F105 V-k appears to be derived from the Humvk325 germline gene and is rearranged with a J-k2 gene. For both chains, the mutational pattern in the rearranged V-H and V-L genes is indicative of an antigen-driven process. These studies show that production of a broadly neutralizing anti-HIV-1 antibody that recognizes determinants within the CD4 recognition sites of the envelope glycoprotein is achieved by rearrangement of the V71-4 and Humvk325 germline variable region genes along with selected individual point mutations in the rearranged genes.

ACCESSION NUMBER: 1993:28788 BIOSIS  
DOCUMENT NUMBER: PREV199395016988  
TITLE: Characterization of the cDNA of a broadly reactive neutralizing human anti-gp120 monoclonal antibody.  
AUTHOR(S): Marasco, Wayne A. [Reprint author]; Bagley, Jessamyn; Zani, Christy; Posner, Marshall; Cavacini, Lisa; **Haseltine, William A.**; Sodroski, Joseph  
CORPORATE SOURCE: Div. Human Retrovirology, Dana-Farber Cancer Inst. 44 Binney St., Boston, Mass. 02115, USA  
SOURCE: Journal of Clinical Investigation, (1992) Vol. 90, No. 4,

pp. 1467-1478.  
CODEN: JCINAO. ISSN: 0021-9738.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 23 Dec 1992  
Last Updated on STN: 23 Dec 1992

=> s albumin fusion protein () shelf life  
L6 3 ALBUMIN FUSION PROTEIN (W) SHELF LIFE

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L6 ANSWER 1 OF 3 USPATFULL on STN

TI Albumin fusion proteins

AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disorders or conditions using albumin fusion proteins of the invention.

ACCESSION NUMBER: 2003:282700 USPATFULL  
TITLE: Albumin fusion proteins  
INVENTOR(S): Ballance, David J., Berwyn, PA, UNITED STATES  
Sleep, Darrell, West Bridgford, UNITED KINGDOM  
Prior, Christopher P., Rosemont, PA, UNITED STATES  
Sadeghi, Homayoun, Doylestown, PA, UNITED STATES  
Turner, Andrew J., Eagleville, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003199043	A1	20031023
APPLICATION INFO.:	US 2001-832501	A1	20010412 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-256931P	20001221 (60)
	US 2000-199384P	20000425 (60)
	US 2000-229358P	20000412 (60)

DOCUMENT TYPE: Utility  
FILE SEGMENT: APPLICATION  
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE,  
ROCKVILLE, MD, 20850  
NUMBER OF CLAIMS: 60  
EXEMPLARY CLAIM: 1  
NUMBER OF DRAWINGS: 18 Drawing Page(s)  
LINE COUNT: 14339

L6 ANSWER 2 OF 3 USPATFULL on STN

TI Albumin fusion proteins

AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disorders or conditions using albumin fusion

proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:244853 USPATFULL  
TITLE: Albumin fusion proteins  
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Sadeghi, Homayoun, Doylestown, PA, UNITED STATES  
Prior, Christopher P., Rosemont, PA, UNITED STATES  
Turner, Andrew J., Eagleville, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003171267	A1	20030911
APPLICATION INFO.:	US 2001-833117	A1	20010412 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-256931P	20001221 (60)
	US 2000-199384P	20000425 (60)
	US 2000-229358P	20000412 (60)

DOCUMENT TYPE: Utility  
FILE SEGMENT: APPLICATION  
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE,  
ROCKVILLE, MD, 20850

NUMBER OF CLAIMS: 59  
EXEMPLARY CLAIM: 1  
NUMBER OF DRAWINGS: 20 Drawing Page(s)  
LINE COUNT: 13208

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 3 OF 3 USPATFULL on STN

TI Albumin fusion proteins

AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disorders or conditions using albumin fusion proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:181414 USPATFULL  
TITLE: Albumin fusion proteins  
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Haseltine, William A., Washington, DC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003125247	A1	20030703
APPLICATION INFO.:	US 2001-833041	A1	20010412 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-256931P	20001221 (60)
	US 2000-199384P	20000425 (60)
	US 2000-229358P	20000412 (60)

DOCUMENT TYPE: Utility  
FILE SEGMENT: APPLICATION  
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE,  
ROCKVILLE, MD, 20850

NUMBER OF CLAIMS: 29



EXEMPLARY CLAIM: 1  
NUMBER OF DRAWINGS: 20 Drawing Page(s)  
LINE COUNT: 15235  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.